# CLINICAL AND CORONARY ANGIOGRAPHIC CORRELATION OF PATIENTS WITH UNSTABLE ANGINA

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# CERTIFICATE

This is to certify that the dissertation entitled **"CLINICAL & CORONARY ANGIOGRAPHIC CORRELATION OF PATIENTS WITH UNSTABLE ANGINA"** is a bonafide work done by **DR.P. JEYASINGH**, Department of Cardiology, Government Rajaji Hospital and Madurai Medical College, Madurai in partial fulfillment of the University rules and regulations for award of DM degree**, Branch II (Cardiology)** under my guidance and supervision during the academic year from **August 2011** to **July 2014.**

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# DECLARATION

I, **Dr. P. JEYASINGH** solemnly declare that the dissertation titled **"CLINICAL & CORONARY ANGIOGRAPHIC CORRELATION OF PATIENTS WITH UNSTABLE ANGINA** has been prepared by me. This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of D.M**., Branch II (Cardiology)** to be held in **August 2014.**

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# CONTENTS

## ABSTRACT

**Introduction :** UA is a clinical syndrome caused by atherosclerotic plaque rupture and thrombosis within a coronary artery . It is defined as angina that is new onset or abruptly increased in intensity ,duration or frequency within the past 60 days. It may present as rest angina,new onset severe angina or increasing angina .Initial evaluation includes risk stratification based on history ,clinical exam , ECG , cardiac enzymes . Among patients with UA who undergo angiogram ,85% will have significant CAD. CABG confers a survival benefit in patients with > 50% LM stenosis or triple vessel disease with LV dysfunction, Importantly patients with no significant lesions at angiography benefit from reorientation of their management.Symptomatic patients with normal coronaries may have significant atherosclerosis by IVUS secondary to coronary artery remodeling

AIM : 1. Risk stratification based on clinical history & presentation, ECG, Enzymes, 2. To Correlate the clinical profile with Coronary angiographic profile 3. To identify the high risk predictors for early intervention

**Materials and methods** *Study design* : Observational and Cross sectional Study*Study population*:Unstable angina patients admitted for coronary angiogram in cardiology ward GRH, Madurai. *Inclusion Criteria* Patients admitted with a history of chest pain diagnosed as unstable angina and subsequently underwent CAG in cardiology ward.

**Results & Conclusion :** Unstable angina commonly affects the age group 45-60yrs in both sexes. 30% of patients in our study was women. Women have normal coronaries compared to men in patients with unstable angina, (30% vs 20%) which suggests a different patho physiological mechanism for their symptoms which leads to difficulty in making a firm diagnosis of UA. Smoking, diabetes, Hyperlipidemia, Hypertension are major risk factors for unstable angina in this study Braunwald class III angina (Rest angina) predicted severity of lesion ( left main & triple vessel disease) in our study. Patients who had High TIMI risk scoring had more severe coronary lesions compared to low TIMI risk score which helps in risk stratification and early intervention.

Significant ST-T changes in ECG predicted more extensive disease which helps in decision making regarding treatment strategy (conservative vs invasive) aVR ST elevation in background of unstable angina predicts left main disease &Triple vessel disease in our study which helps risk stratification and early intervention. ECHO evidence of LV dysfunction predicted Triple vessel disease /LM disease. Out of the 100 pts who underwent coronary angiogram in our study 27 pts had Single Vessel disease ( type A lesions predominantly) 24 pts had two Vessel disease .( type B lesions predominantly) 26% had three vessel disease. (type B lesions predominantly) 14 patients had Left Main Coronary artery disease. 23 patients had normal or insignificant coronary artery lesions. 9 patients had thrombus containing lesion who had rest angina, out of whom 6 patients had SVD and 3 patients had multivessel disease. 3 patients had total occlusion with TIMI '0' flow.

# INTRODUCTION

Acute Coronary Syndrome is a useful practical term for referring to any pattern of clinical symptoms that is consistent with acute myocardial ischemia. Two closely related forms of ACS – Unstable angina UA and NSTEMI. Unstable angina and the closely related condition NSTEMI are very common manifestations of CAD. Previously UA and NSTEMI were considered as separate entity but pathophysiological mechanism for both involves the rupture or erosion of atherosclerotic plaque with subsequent thrombus formation that significantly obstruct the coronary artery lumen There is a complex overlap between the two syndromes. Accordingly patients with either of these syndromes are frequently treated identically with individual variations in a management depending on a classification of high, intermediate and low risk.

They differ primarily in whether the ischemia is severe enough to cause sufficient myocardial damage to release detectable quantities of a marker of myocardial injury [troponon I, troponin T, etc]. Recently, many new information has come to light concerning the diagnosis and subsequent management of patients with either of these two types of ACS. It came to be recognized that the active plaque was not unique suggesting a more diffuse inflammatory disease. These concepts led to a

new era of research in cell biology and clinical investigation supported by emerging technologies and procedurally sophisticated clinical research, providing a basis for our current evidence based approaches to therapy. Every year in the United States, ACS is responsible, either as a primary or secondary diagnosis, for approximately 1.36 million hospitalizations of which about 0.81 million are for MI and the remaining for Unstable Angina.

**DEFINITION**

The definition of unstable angina is largely based on clinical presentation. Stable angina pectoris typically manifests as a deep ,poorly localized chest or arm discomfort which is precipitated by physical exertion or emotional stress, and relieved within 5- 15 min by rest and or by sublingual nitroglycerin. In contrast Unstable angina is defined as angina pectoris [or equivalent type of ischemic discomfort] with at least one of the three features.

1. Occurring at rest or minimal exertion and usually lasting > 20 min[ if not interrupted with nitroglycerine administration
2. Being severe and described as frank pain and of new onset [within 1 month]
3. Occurring with a crescendo pattern [ more severe, prolonged or frequent than previously]

Of this group, approximately one half will have proof of myocardial necrosis on the basis of elevated serum cardiac markers, such as creatinine kinase isoenzyme CK-MB and or troponin –I or T and thus confirming a diagnosis of NSTEMI

## CLASSIFICATION

AS UA/NSTEMI comprises a broad group of patients, several classification schemes have been proposed. The Braunwald clinical classification: In this classification, patients of UA/NSTEMI are divided into three groups.

**Primary UA :** Secondary UA [eg. With angina related to obvious precipitating factors , such as anaemia]

**Post MI UA : Class 111 angina** [angina at rest within 48 hrs 10.8%

This classification predicts coronary thrombus at angiography ,or in atherectomy specimens.

**Classification of angina according to severity of ischemia**

***Braunwald clinical classification of Unstable Angina / NSTEMI***

| **Type/ class and definition** | **Death or MI at 1 year** |
|---|---|
| **Primary angina**[ in absence of extracardiac condition] | 8.5% |
| **Secondary angina** [ presence of extracardiac condition ] | 14.1 % |
| **Post MI angina** [ Within 2 wks after AMI ] | 18.5% |
| **Class 1 angina**[new onset of angina , no rest pain ] | 7.3% |
| **Class 11 angina**[angina at rest within past month] | 10.3% |

**Classification according to etiology**

- Five pathophysiological approaches have been proposed
- Plaque rupture or erosion with superimposed non occlusive thrombus ( by far the most common cause of UA/NSTEMI)
- Dynamic obstruction ( ie , coronary spasm )
- Progressive mechanical obstruction
- Inflammation

**Secondary Unstable angina ( MVO2)**

As noted subsequently, several new serum markers can serve as effective tools in identifying these pathophysiological processes and in predicting outcome thus forming the foundation of a multimarker strategy for evaluation and risk stratification

**PATHOPHYSIOLOGY**

Although traditionally, attention has been focused only on the acute phase of UA/NSTEMI , the pathophysiology may actually have its roots several decades before the actual clinical event and this may span more than 20 yrs at times. The acute event, which usually involves thrombus formation at the site of ruptured or eroded atherosclerotic plaque ,is currently referred to as atherothrombosis, a term that is replacing atherosclerosis because it more aptly describes the pathophysiology of the disease that involves both progression and

disruption of atheroma and superimposed thrombosis. The acute ischemia can also originate from an increase in myocardial oxygen demand (eg. Precipitation by tachycardia or hypertension) and/or by reduction in supply (eg. due to reduction in coronary lumen diameter by platelet rich thrombi, vasospasm , or hypotension)

**PLAQUE DISRUPTION AND EROSION**

Many of the mechanical cellular and molecular factors contributing to plaque disruption have been elucidated in recent years Factors that modulate the development and complications of ACS are as follows

Location of culprit coronary lesion
Stenosis morphology and severity
Extent of plaque rupture or erosion
Inflammatory substrate
Endothelial function
Degree of coronary vasoconstriction
Microembolisation and microvascular obstruction
Extent of collaterals
Thrombotic factors

- Platelet aggregability and reaction
- Leukocyte activation
- Intrinsic clotting activity
- Plaque tissue factor level
- Level of fibrinolytic activity, Blood viscosity

**Systemic factors**

Heart rate and blood pressure catecholamine levels (smokes, cocaine and stress) ,blood lipid levels

Plaque rupture most commonly takes place at the shoulder region of the plaque, where the plaque joins the adjacent vessels wall ; this area of the plaque is commonly infiltrated with inflammatory cells and subject to high sheer forces.

Vulnerable plaques or high risk plaques tend to have a thin fibrous cap and a large lipid pool which influence the biomechanical properties of the plaque and increase the likelihood of rupture. Conversely, fibrosis and calcification appear to attenuate the risk of rupture.

Erosion occurs centrally through the cap rather than at the plaque shoulders. Erosion appears to be more common among women who smoke, whereas plaque rupture occurs more frequently in hyperlipidemic men.

**INFLAMMATION**

It plays the central role in plaque disruption. Macrophages and T lymphocytes accumulate in atherothrombotic plaque because of the expression of adhesion molecules on monocytes, endothelial cells, and leukocytes. They release proinflammatory cytokines and chemokines as well as matrix metalloproteinases which include collagenases and

gelatinases, are released from macrophages and degrade the collagen that provide strength to the fibrous cap. Basically, inflammatory stimulus causes a biochemical storm within the high risk plaque, leading to rupture of its fibrous cap. It is now believed that atleast in some individuals with UA/NSTEMI ,inflammation may be a much more widespread process. The clinical manifestations of this phenomenon are now recognized to include the occurrence of multiple. simultaneous complex coronary plaques at the time of presentation in some patients with UA/NSTEMI

**INFECTION**

Chlamydia pneumonia, CMV and helicobacter pylori have been identified within human atherosclerotic lesions. Furthermore, antibodies against Chlamydia, heat shock proteins can cross react against heat shock protein produced by endothelium, resulting in endothelial damage and accelerated atherosclerosis.

However these associations do not indicate causality and antibiotic regimens against Chlamydia have not demonstrated any conclusive benefit

**PLATELET AGGREGATION**

Platelets play a key role in the transformation of a stable atherosclerotic plaque to an unstable lesion with three major steps in the process:

1, Adhesion

2, Activation

3, Aggregation

Following disruption of an atherosclerotic plaque, the subendothelial matrix (collagen and tissue factor) is exposed to circulating blood, leading to platelet adhesion via the platelet GPIb receptors through its interaction with VWF and GPVI binding to collagen

The next step is platelet activation which has 3 components

I, Change of shape of platelet from discoid to speculated

2,Release of platelet content including thromboxane A2 ( TXA2)

3, Activation of GP IIb / IIIa on their surface

The next step is platelet aggregation in which platelet plug is formed by cross linking of platelet by fibrinogen ( or von Willebrand factor )

**CLOTTING CASCADE** ( Secondary Hemostasis )

Release of tissue factor appears to be the key mechanism of initiating hemostasis following plaque rupture. This leads to factor X being activated to factor Xa which in turn generates thrombin. This thrombin converts fibrinogen into fibrin, activates factor XIII which stabilizes the fibrin clot and stimulates platelet aggregation ,which can further propagate thrombus.

**THROMBOSIS**

It also has a great role to play in the pathogenesis of UA/NSTEMI. Coronary thrombi have been visualized at the site of a ruptured plaque in autopsy studies, in coronary atherectomy specimens and with coronary angioscopy

**CORONARY VASOCONSTRICTION**

Culprit lesions in UA/NSTEMI demonstrate an increased response to vasoconstrictor stimuli compared to other coronary artery segments or culprit lesions of patients with stable angina .Intense focal spasm of a segment of an epicardial coronary artery occurs in prinzmetal variant angina. There is increasing recognition of the role of coronary vasoconstriction causing microcirculatory angina resulting from constriction of the small intramural coronary resistance vessels.

## SECONDARY UNSTABLE ANGINA

This occurs in patient of CAD due to imbalance between myocardial oxygen supply and demand which is precipitated by conditions outside the coronary tree Increased oxygen demand occurs in tachycardia, fever, thyrotoxicosis, hypertension and aortic stenosis.

Reduced oxygen supply results from anaemia, hypotension, and hypoxemia (due to pneumonia or CHF )

## PROGRESSIVE MECHANICAL OBSTRUCTION

UA/NSTEMI can also occur from progressive luminal narrowing of the coronary artery. This is most commonly seen in the setting of restenosis following PCI. However, this may occur without PCI and the mechanism appears to be related to rapid cellular proliferation.

## NATURAL HISTORY OF UNSTABLE ANGINA

Unstable angina patients have low short-term mortality (1.5-2%) from 0-30 days of presentation than patients with NSTEMI or STEMI .The early mortality risk of the 2 types of MI is same (3-5%). The early mortality risk in UA relates to the extent of myocardial damage and hemodynamic compromise and is less than STEMI. In contrast the long term outcome –for mortality and nonfatal events is worse for patients UA compared with STEMI .This finding results from recurrence of ACS

in pts with UA as well as old age ,great extent of CAD,prior MI and comorbidities like diabetes & renal dysfunction.

**CLINICAL PRESENTATION**

Pain is the main symptom in patients with UA/NSTEMI. It is generally located to the substernal region but it may involve jaw, neck, shoulder, arm back or epigastrium. Some patients present with unexplained dyspnea, fatigue, palpitation, nausea, vomiting and diaphoresis which are called angina equivalent. Such atypical presentations are more common in older adults and women. Rarely patient may present with syncope as primary symptoms. Features which are not usually suggestive myocardial ischemia are sharp stabbing pleuritic pain, reproduction of pain on palpation or with movement, very brief episodes of pain that lasts a few seconds or less, pain that may be localized at the tip of finger, particularly over the LV apex or costochondral junction Although typical characteristics substantially increase the probability of CAD, atypical features do not totally exclude the possibility of ACS. The chest pain that is relieved by sublingual nitroglycerine does not always predict ACS.

In younger patients without any CAD risk factors, one should enquire about cocaine and methamphetamine addiction as these agents

cause coronary vasospasm and thrombosis. Urine toxicology should be obtained in such patients

**DEMOGRAPHICS**

Apart from nature of anginal symptoms prior history of CAD, sex, age and the number of traditional risk factors present are related to likelihood of ischemia due to CAD. Prior history of MI has been associated with high likelihood of multivessel CAD. Gender appears to have an impact on prevalence of ACS. Women comprise 30-45% of patients with UA, compared to approximately 25-30% of patients with NSTEMI and 20% of patients with STEMI. In addition, compared with STEMI, patients with UA/NSTEMI also have higher rate of prior MI, angina, previous coronary revascularization, and extracardiac vascular disease. Older patients have more risk of multivessel CAD and adverse outcome. The slope of increased risk is steepest beyond age 70 yrs . The presence of conventional risk factors like hypertension, hypercholesterolemia, cigarette smoking have limited utility in predicting the ischemic symptoms but does relate to adverse prognosis. Other risk factors like diabetes, family history of premature CAD and presence of extracardiac vascular disease also have significant prognostic and therapeutic implications.

## PHYSICAL EXAMINATION

The main objectives of physical examination are as follows:

To unmask potential precipitating causes of myocardial ischemia (eg. Uncontrolled hypertension, thyrotoxicosis, or GI bleeding )

To identify associated comorbid conditions that could relate to therapeutic risk and decision making (pulmonary disease and malignancy)

To rule out alternate diagnosis eg. Unequal pulse may suggest aortic dissection, pericardial friction rub may suggest acute pericarditis, pulsus paradoxus indicates cardiac tamponade Large areas of ischemia are associated with diaphoresis, pale cool skin, sinus tachycardia,a third or fourth heart sound ,basilar rales and hypotension.

## DIAGNOSTIC TOOLS

Apart from history and clinical examination, the following are useful:

Electrocardiogram

Echocardiogram and noninvasive myocardial imaging

Biomechanical markers

Imaging of coronary anatomy

## ELECROCARDIOGRAM

It is the first line diagnostic tool in the evaluation of patients with suspected Non ST Elevation acute coronary syndrome (NSTE-ACS). It should be obtained within 10 min after first medical contact. ECG not only strengthens support to diagnosis of UA/NSTEMI but also helps in risk stratification based on pattern and magnitude of abnormalities. In UA,ST depression, transient ST elevation, and or T-wave inversion occur in 30 – 50% of patients, depending on the severity of clinical presentation. Several analysis have shown that new ST segment deviation, even only 0.05 mV, is specific and important measure of ischemia and prognosis. T –wave changes are sensitive for ischemia, but are less specific, inless they are marked ( > 0.3 mV). Conversely, small changes in T waves with inversions of only 0.1 mV, appear to add little to clinical history. ST depression of > 1 mm is associated with 11% rate of death and MI at 1 yr. ST depression of > 2 mm implies about a six fold increased mortality risk. Deep symmetrical inversion of the T waves in the anterior chest leads is often secondary to a significant stenosis of the proximal left anterior descending coronary artery . A completely normal ECG in a patient with chest pain does not rule out the possibility of ACS, because a small percentage of such patients are eventually found to have NSTEMI.

## CONTINUOUS ECG MONITORING

The standard ECG at rest may not adequately reflect the dynamic nature of coronary thrombosis and myocardial ischemia. Almost two-thirds of all ischemic episode, in the phase of instability, are clinically silent and hence not likely to be evident on conventional ECG. On line continuous computerized 12 lead ST segment monitoring is a useful diagnostic tool. Several studies have shown that 15-30% of patients with NSTE- ACS have transient episodes of ST- segment depression. These patients tend to have an increased risk of subsequent cardiac events. It also reveals arrhythmias in association with an acute episode and recurrent ischemia which may be clinically silent.

## EXERCISE OR OTHER STRESS TESTING

In patients who continue to have typical ischemic chest pain, no stress test should be performed in the acute phase .However ,in patients with nondiagnostic with nondiagnostic ECG, stress test has a predictive value provided there is no pain , no signs of heart failure and normal biomarkers (repeat testing). A negative exercise testing has a high negative predictive value.

## ECHOCARDIOGRAM AND NONINVASIVE MYOCARDIAL IMAGING

Experienced hands can detect transient localized hypokinesia or akinesia in segments of the left ventricle wall during ischemia, with normal wall motion on resolution of ischemia. Furthermore, differential diagnosis such as aortic stenosis, aortic dissection, pulmonary embolism or HOCM can be excluded. LV systolic function is an important prognostic marker in patients with ischemic heart disease and can be easily and reliably assessed by echocardiography.

Similarly, stress scintigraphy or magnetic resonance imaging may be used :if available. MRI is useful to assess myocardial viability .Rest myocardial scintigraphy has been shown to be helpful for initial triage of cases presenting with chest pain without ECG changes or evidence of ongoing MI.

## BIOMECHANICAL MARKERS

The elevation of markers of myocardial necrosis [ie creatinine kinase MB ], troponin T or I identifies patients with the diagnosis of NSTEMI. Microembolization of platelet aggregates and components of the ruptured plaque are believed to be responsible for the release of myocardial markers in many of these patients. A cardiac specific troponin is the preferred marker because it is more specific than CK-

MB and identifies a greater percentage of patients presenting with NSTEMI. Patients with negative cardiac markers within 6 hrs of the onset of symptoms that are consistent with ACS, should have biomarkers remeasured in the timeframe 8- 12 h after symptom onset.

The diagnosis of NSTE-ACS should never be made, however, only on the basis of cardiac biomarkers. Elevation of biomarkers should be interpreted in the context of the clinical finding

**IMAGING OF CORONARY ANATOMY**

Imaging modalities provide extremely valuable information on the presence and the severity of CAD. The gold standard is still conventional invasive coronary angiography. Angiographic assessment of the characteristics and location of the culprit lesion as well as other lesions is essential if revascularization is considered. Cardiac computed tomography ( CT- angio ) cannot be recommended as the coronary imaging modality of choice at present. Role of MRI as an imaging tool for coronary arteries is even less established.

**OTHER LABORATORY TEST**

A chest x-ray is generally performed on admission to screen for pulmonary congestion, a finding that is associated with an adverse prognosis. Since serum cholesterol level falls by as much as 30- 40 %

beginning 24 h following ACS, they should be measured at the time of initial presentation

**DIAGNOSTIC PATHWAYS IN THE EMERGENCY DEPARTMENT**

The current emergency department [ED] pathways for diagnosis of UA/NSTEMI utilize four major diagnostic tools: Clinical history , ECG,cardiac markers, and stress testing. Initial information is integrated to assign patients with chest pain into one of four categories

1, a noncardiac diagnosis

2, Chronic stable angina

3, Possible ACS

4, Definite ACS

**RISK STRATIFICATION**

All patients with chest discomfort or other symptoms suggestive of an ACS should be subjected to rapid clinical evaluation of the likelihood of the risk of obstructive CAD ( high, intermediate, or low ) and early risk stratification for the patient management. Early risk assessment is done by actual finding from history, clinical examination, ECG, cardiac biomarkers, and renal function measurement

Risk assessment is necessary in I, selection of site of care [CCU, monitored step down unit, or outpatient setting] ii, therapeutic decision

including platelet GP II b/III a inhibitors and early invasive therapy iii, prognostic assessment. However optimal risk assessment should involve simultaneous multivariate analysis of multiple risk factors that are present in a given individual. There are three such risk assessment protocols, namely TIMI, GRACE and PURSUIT risk scores, are available for above mentioned purpose.

## RISK STRATIFICATION IN UA/NSTEMI

The 2011 ACC/AHA Guidelines for the Management of patients with unstable angina and NSTEMI recommend that all patients with chest discomfort or other symptoms suggestive of an ACS should undergo a rapid clinical determination of the likelihood risk of obstructive CAD (ie high, intermediate or low) and early risk stratification and results should be implemented in patient management . Basis of early risk stratification centers around history, physical examination, ECG, cardiac biomarker, and renal function measurement. The spectrum of risk in ACS ranges from 30 day mortality of 1.7 % for patients with UA, 7.4% for NSTEMI and 11.11% for STEMI, based on data from a global registry.

**MULTIVARIATE RISK ASSESSMENT SCORES FOR NSTEMI**

Integrating all the factors mentioned above, several groups have developed comprehensive risk scores. Of these, TIMI risk score is the one most popularly used. The TIMI risk score identified seven independent risk factors:

1, age > 65 yrs

2, > 3 risk factors for CAD

3, documented CAD at catheterization

4, ST deviation > 0.5 mm

5, > 2 episodes of angina in the last 24 hrs

6, aspirin use within the prior week

7, elevated cardiac markers

Appropriate utilization of TIMI risk score can risk-stratify patients o NSTE-ACS across a 10 fold gradient of risk from 4.7% to 40.9 %( $P<0.001$).The TIMI risk score was validated internally within the TIMI 11 B trial and two separate cohorts of patients from the ESSENCE trial. Utility of the TIMI risk score lies mainly in the fact that higher TIMI risk scores benefit more from potent and invasive therapies, ie, enoxiparin ( in comparison to unfractionated heparin ), GP IIb/IIIa inhibitor( in comparison to placebo) and early invasive strategy( in comparison to conservative strategy)

Another well known system is the GRACE risk model. It was developed on the basis of 11,389 patients in the GRACE registry and validated in the GUSTO –II b cohort. It predicts in-hospital mortality in the entire spectrum of ACS, ie STEMI, NSTEMI,UA . The eight variables used in the GRACE risk model include

1, older age 2, Killips class 3, systolic blood pressure 4, ST-segment deviation 5, Cardiac arrest during presentation 6,Serum Creatinine level 7, Positive initial cardiac biomarkers and 8, Heart rate. All these variables cause mortality during the period from hospital discharge to 6 months and the total score can be determined by the application of the sum of scores to a reference nomogram.

Another risk model ( the PURSUIT risk model ) was developed on the basis of the PURSUIT trial and it is another useful tool to predict the 30-day incidence of death and composite of death or MI. The markers included in the risk model( in order of strength) are 1, age 2, heart rate 3, systolic blood pressure 4,ST segment depression 5, signs of heart failure, and 6, cardiac biomarkers.

All three risk scores (TIMI, GRACE, and PURSUIT ) demonstrated good predictive accuracy for death and MI at 1 yr and they help us immensely in identifying those patients of NSTE-ACS who are likely to benefit from aggressive therapy, including early invasive

strategy. However, with the emergence of new biomarkers, it is likely that these too will be included in comprehensive risk scores in future, after they become established in clinical practice.

**RISK STRATIFICATION**

| **TIMI SCORE** | | |
|---|---|---|
| | 0-2 points | Low risk |
| | 3-4 points | Intermediate risk |
| | 5-7 points | High risk |
| **GRACE RISK SCORE** | **Inhospital risk stratification** | |
| <108 | Low | |
| 109-140 | intermediate | |
| >140 | High | |
| **GRACE RISK SCORE** | **Predischarge to 6 months risk stratification** | |
| <108 | Low | |
| 89-118 | Intermediate | |
| >118 | High | |

The extent of coronary disease among patients with UA/NSTEMI enrolled in the invasive arm of TACTICS-TIMI 18, who systematically underwent angiography, was: 34 percent had significant obstruction (>50 percent luminal diameter stenosis) of three vessels; 28 percent had two vessel disease; 26 percent had single vessel disease; and 13 percent

had no coronary stenosis >50 percent. Approximately 5 to 10 percent had left main stem stenosis >50 percent. Registries of unselected UA/NSTEMI patients have reported similar findings. Women and non-whites with UA/NSTEMI have less extensive coronary disease than their counterparts, whereas patients with NSTEMI have more extensive disease than those who present with unstable angina.

Women and non-whites comprise a larger proportion of patients with symptoms of UA/NSTEMI without epicardial coronary disease-suggesting either a difficulty in making a firm diagnosis of UA/NSTEMI in these groups and/or a different pathophysiological mechanism for their clinical presentation  Approximately one third of patients with UA/NSTEMI without a critical epicardial obstruction have impaired coronary flow assessed angiographically-suggesting a pathophysiological role for coronary microvascular dysfunction. The short-term prognosis is excellent in this group of patients.

The culprit lesion in UA/NSTEMI typically exhibits an eccentric stenosis with scalloped or overhanging edges and a narrow neck. These angiographic findings may represent disrupted atherosclerotic plaque, thrombus, or a combination. Features suggesting thrombus include globular intraluminal masses with a rounded or polypoid shape. “Haziness” of a lesion has been used as an angiographic marker of

possible thrombus, but this finding is less specific. Patients with angiographically visualized thrombus have impaired coronary flow and worse clinical outcomes, compared to those without thrombus. Patients with UA/NSTEMI have impaired coronary flow as measured by the TIMI flow grade or frame count, and TIMI myocardial perfusion grade—especially those with an elevated troponin level, which is independently associated with adverse outcomes.

ACC/AHA Recommendations for coronary Arteriography in patients with Unstable Angina/Non-ST Elevation MI

| Class | Recommendation | LOE |
|---|---|---|
| Class I | Early Invasive Strategy | A |
| | Recurrent angina inspite of intensive therapy | |
| | Elevated troponin level | |
| | New ST segment depression | |
| | CHF symptoms | |
| | Noninvasive stress test –high risk finding | |
| | Depressed LV systolic function EF <40 | |
| | Hemodynamic instability | |
| | Sustained Ventricular Tachycardia | |
| | PCI within 6 months | |
| | Prior CABG | |

In absence of these findings, either early conservative / invasive BClass IIa Early invasive therapy in pts with repeated presentations for ACS C Class III Patients with extensive comorbidities

Acute chest pain and low likelihood of ACS

Patients who will not consent to revascularization

**TABLE 53-2 -- Clinical Indicators of Increased Risk in UA/NSTEMI**

| **History** |
| --- |
| Advanced age(>70yr)<br>Diabetesmellitus<br>Post–myocardialinfarctionangina<br>Prior peripheral vascular disease<br>Prior cerebrovascular disease |
| **Clinical Presentation** |
| Braunwald class II or III (acute or subacute rest pain)<br>Braunwald class B (secondary unstable angina)<br>Heart failure/hypotension<br>Multiple episodes of pain within 24 hr |

| **ECG** |
| --- |
| ST segment deviation ≥0.05 mV<br><br>T wave inversion ≥0.3 mV<br><br>Left bundle branch block |
| **Cardiac Markers** |
| Increased troponin T or I or creatine kinase-MB<br><br>Increased C-reactive protein or white blood cell count<br><br>Increased B-type natriuretic peptide<br><br>Elevated creatinine<br><br>Elevated glucose or hemoglobin $A_1C$ |
| **Angiogram** |
| Thrombus<br><br>Multivessel disease<br><br>Left ventricular dysfunction<br><br>UA/NSTEMI = unstable angina/non-ST elevation myocardial infarction. |

## TREATMENT

### General Measures

Based on the risk of future events ,patients with a presumed diagnosis of UA/NSTEMI may be admitted to a coronary care unit(high risk),a telemetry unit ( intermediate or low risk) or may be managed in a chest pain unit (low risk),Continuous telemetry monitoring should be available to detect dysrhythmias. Even though not rigorously studied .oxygen supplementation is frequently used to provide adequate supply of oxygen, especially to the patients with oxygen saturation of 90% or below.

### Aim of therapy

Relieve ischaemia

Control symptoms

Prevent Complications including recurrent episodes of myocardial ischemia/necrosis

B –blockers,nitrates and calcium channel blockers reduce the risk of recurrent ischemia .

Angiotensin converting enzyme (ACE ) inhibitors .In patients with LV dysfunction ,ACE inhibitors improve survival and ventricular remodelling

Revascularisation eliminates ischemia in many patients

The risk of progression to MI ,or recurrent MI is diminished by antiplatelet and antithrombotic drugs and by invasive treatment of the culprit lesion . Aggressive statin therapy plays an increasing important role after ACS

Initial conservative versus initial invasive strategy

Two approaches to managing patients with UA have evolved .Based on the host of factors including an overall assessment of patient risk , a decision to pursue either an early invasive strategy or an initial conservative strategy needs to be made early. Those pts selected for conservative approach are managed with optimal medical therapy and CAG only in selected symptoms

Hospitalised patients with non-ST elevation and ACS pts should be treated with ASA, clopidogrel or Prasugrel, antithrombotic therapy, a beta blocker and statin.

In selected high risk patients a GPIIb/IIIa inhibitor may be indicated . Furthermore .critical decisions are required regarding the angiographic strategy.

One option ,commonly termed the early invasive approach ,incorporates an angiographic approach in which patient undergoes coronary angiography within 24 to 48 hrs and revascularization is performed if suitable coronary anatomy is identified. The alternative approach ,the early conservative approach is guided by myocardial ischemia, with angiography reserved for patients with recurrent

ischemia at rest or findings on a predischarge noninvasive evaluation for ischemia that do not support low risk status. This strategy is better described as selective invasive because it requires aggressive medical intervention and risk stratification.

Regardless of angiographic strategy used ,an assessment of LV function should be strongly considered because it caries prognostic information and it is imperative to treat patients who have impaired LV systolic function with both ACE inhibitors and beta blockers .In appropriate candidates CABG surgery has to be done.

Randomized trials that have compared an early invasive versus an early conservative approach in different patient population

TIMI III B , VANQISH trials – Both of these trials showed similar long term outcomes between early invasive and conservative treatment strategies, however, there was an increase in early mortality associated with invasive therapy in the VANQWISH study

FRISC II trial – pts with UA were randomized in a factorial design to an early invasive or conservative strategy and to dalteparin or placebo. An early invasive strategy was associated with a reduction in rate of death or MI at 6 months (9.4% vs 12.1%,p=0.031) and reduced symptom of angina and rehospitalisation, regardless of treatment with dalteparin.

**Hospital Discharge and Post discharge care**

The risk of progression to MI or the development of recurrent MI or death is highest during the first two months after UA/NSTEMI. Thus although patients with UA usually receive definite treatment during hospitalization ,close followup care after hospital discharge is imperative

Specific recommendations regarding a secondary prevention postdischarge medication regimen are listed below

Anti ischaemic medicines – nitrates

Aspirin 75-162 mg

Thienopyridine therapy

B blockers

ACE inhibitors

Aldosterone receptor antagonist - EPHESUS

Statin therapy – MIRACL, PROVE IT –TIMI 22, ARMYDA-ACS trials

## AIM

- **Risk stratification based on clinical history & presentation, ECG, Enzymes**

- **To Correlate the clinical profile with Coronary angiographic profile**

- **To identify the high risk predictors for early intervention**

# METHODOLOGY

## Study subjects

100 patients admitted in intensive care unit of department of cardiology, Govt. Rajaji Hospital Madurai, clinically diagnosed as unstable angina were studied. This is a prospective study

Risk stratification was made clinically, ECG, Enzymes, TIMI RISK score and all these patients were subjected to coronary angiogram.

This study was a cross sectional analytical study planned from December 2013 to May 2014

## CLINICAL EXAMINATION

Patients were risk stratified into low/intermediate /high risk based on Braunwalds classification – class I, 2 ,3

TIMI Risk score was done for all patients by including 7 parameters

1, age>65

2, >3 CAD risk factors (high lipids, F/h, HTN, DM, Smoking)

3, Prior coronary stenosis >50%

4, Aspirin in last 7 days

5, > 2 anginal events in <24 h

6, ST segment deviation

7, Elevated cardiac markers

Total score of 0-7/7 was given

**ELECTROCARDIOGRAM**

12 lead ECG was done for all patients at the time of admission, depending on the ECG changes ( normal or unchanged ECG , T wave inversion >0.2 mV, ST-segment changes) the patients were risk stratified into low, intermediate and high risk

**CARDIAC MARKERS**

Qualitative Troponin tests were used to risk stratify into low,intermediate and high risk categories

**ECHOCARDIOGRAPHY**

Philips IEE 33 cardiac ultrasonography with S4 low frequency phased array transducer with TDI capabilities was used for this study – LV function was assessed using Teichoz method and eyeballing. Segmental distribution of Regional wall motion abnormality was studied

**CORONARY ANGIOGRAPHY**

All patients underwent coronary angiogram through femoral or radial access using judkins technique

The procedure was performed within 72 hrs of admission to hospital . All obstructive lesions were visualized in multiple planes . Qualitative morphological analysis of all angiograms are performed . In each case we attempted to identify the ischemia related artery and a culprit lesion with a visual diameter stenosis of > 70 % on the basis of

anatomy ( LAD, LCx, RCA lesions), 50% for LMCA or LM equivalent lesions which was taken as significant anatomical stenosis

All the lesions are characterized into Type A (high success, >85%; low risk), Type B lesions (moderate success, 60% - 85%; moderate risk) Type C lesions (low success, <60%; high risk) according to ACC/AHA classification

EXCLUSION CRITERIA

1. Doesn't opt for inclusion in the study
2. Chronic renal insufficiency GFR <60ml/min/m2
3. History of allergy
4. STEMI / CSA

**Statistical Analysis :**

The information collected regarding all the selected cases were recorded in a master chart. Data analysis was done with the help of computer by using SPSS software and Sigma Stat 3.5 version (2012). Using this software, frequencies, percentage, mean, standard deviation and ‘p’ value were calculated through Chi square and P value of $< 0.05$ was taken as significant.

# REVIEW OF LITERATURE

Among patients with stable CAD ,risk is proportional to the number of vessels with greater than 50% diameter stenosis and the presence and severity of LV dysfunction .However the relative prognostic impact of the extend of CAD is probably less with ACS because the risk of short term events is dominated by features of the culprit lesion ,such as whether it induces ST segment depression or troponin release.

Among patients with UA/NSTEMI who undergo arteriography, approximately 25% had one vessel disease, 25% had two vessel disease ,and 25 % had three vessel disease. Ten percent have significant main stenosis and the other 15 % will have coronary luminal narrowing of <50% or normal vessels on arteriography. Patients with left main stenosis of at least 50%, or three vessel disease with LV dysfunction, derive a survival benefit from coronary artery bypass surgery compared medical therapy .Importantly ,patients with no significant lesions at angiography benefit from a reorientation of their management. Noncardiac causes of chest pain should be considered (including pulmonary embolism), as well as syndrome X and variant angina .If the coronaries are completely angiographically normal, antithrombotic and

antiplatelet drugs can often be discontinued and the need for antianginal medication reassessed

Patients who are most likely to have no significant lesion at angiography tend to be women with no ECG changes. Nevertheless, the finding of no significant lesions at angiography is usually unanticipated .Importantly ,symptomatic patients without significant obstructive CAD seen with angiography may have more severe atherosclerosis detected by IVUS caused by eccentric coronary artery remodeling ,which preserves the lumen size .Therefore in selected patients, more invasive testing at the at the time of coronary angiography, including use of IVUS or measurement of coronary flow reserve using adenosine as a vasodilating agent ,may be indicated to help better establish the cause of the acute chest pain

The following studies correlated the clinical and coronary angiographic profile in unstable angina

**1, Correlation of angiographic morphology and clinical correlation JACC 1997 volume .29 pg 519-525**

A prospective study of 284 patients with UA who underwent cardiac catheterization. A single angiographer with no knowledge of the clinical classifications interpreted all angiograms. Culprit lesions identified in 200 patients were classified as simple or complex. Complex

lesion includes complex morphology, intracoronary thrombus or total occlusion, Lesions were quantitatively assessed and TIMI flow was assessed. Univariate and multivariate logistic regression analysis of the angiographic findings were performed controlling for all cardiac risk factors, previous angioplasty, or bypass surgery and multivessel disease and we sequentially compared Braunwald class 111,C and c with classes < 3 , <C , <c respectively

Results: Class 111 was associated with complex lesions and decreased TIMI flow, Class C angina correlated with complex lesions and decreased TIMI flow, ICT. The degree of stenosis by QCA was not associated with any particular Braunwald class : Conclusions: Recent rest angina, and refractory or postinfarction angina or both are strongly associated with the general category of complex lesions and specifically with angiographically detected ICT and decreased TIMI 3 flow.

**2, Correlation of Braunwalds clinical classification of Unstable angina pectoris with angiographic extent of disease, lesion morphology and intraluminal thrombus Indian Heart Journal ,1998 May-Jun; 50(3): 300-6**

100 consecutive patients (81 male and 19 female) with unstable angina undergoing coronary angiogram were divided according to

Braunwalds clinical classification. Seventeen patients had new onset angina[class 1] 68 had subacute angina [class 11] 15 had class 111 rest angina . 27 patients had secondary unstable angina [class A] 49 had primary UA [class B] 24 patients had postinfarction unstable angina [class C]. ST- T changes on ECG were present in 54[54%] while absent in 46[46%].Single vessel disease seen higher in class 1 as compared to class 11 and 111. Single vessel disease was highest in class C as compared to class B. Double vessel disease was higher in class B as compared to class A. Triple vessel disease incidence was not found to be significantly different among different clinical classes. Morphology of coronary artery lesions was classified according Ambrose classification. Concentric lesions were found to be higher in class C as compared to class B.Statistically significant differences was not present in the distribution of other morphological type of lesions among different clinical classes. In the whole study group only ICT was found in 17 % of patients

3, **C-reactive protein concentration and angiographic characteristics of coronary lesions**

In a cross sectional study, we examined 103 consecutive patients undergoing cardiac catheterization for suspected CAD. We assessed the association of preprocedural CRP concentrations with clinical

presentations [unstable angina ] and angiographic features of coronary lesions. twenty patients had unstable angina. Independent predictors of unstable angina included increased CRP OR,2.93 per 10 fold increase in CRP,95% confidence interval. Thirty two culprit lesion had macroscopic thrombus or eccentric/irregular discrete morphology without total occlusion. Increased CRP was the strongest predictor of such features and the effect was independent of the presence of unstable angina

**4,Correlation of ECG changes with coronary angiographic morphology in patients presented with rest pain**

**University Heart Journal 2007:3:57-59**

The study was performed to see the correlation of ECG changes with CAG findings in patients of unstable angina presented with prolonged rest pain. A total of 30 cases were taken and was divided into 3 groups according to their ST segment and T wave changes in ECG. Echocardiogram was done and selective coronary angiogram was done within 5 -15 days of presentation. The study showed that patient with significant ECG change had more extensive coronary artery involvement than the patients with less significant ECG changes or normal ECG with better preserved ejection fraction. So ECG changes

can give a clue about severity, long term prognosis and outcome of disease.

**5, ST depression with negative T waves in leads V4-V5 –A marker of severe CAD in NSTEMI: A prospective study of Angina at rest, with troponin, clinical, electrocardiographic and angiographic correlation. Annals of noninvasive electrocardiography Vol 9, issue 3, pages 207-214, July 2004**

We studied the correlation of ST-T changes in 12 lead ECG recorded during pain, to clinical and angiographic findings and in hospital prognosis in patients with NSTEMI and elevated troponin levels. 50 consecutive could be differentiated into 2 groups [1] 25 pts with ST segment depression and a negative T wave maximally in leads V4-5 [2] 25 patients with ST segment depression and a positive T wave in precordial leads with maximal ST depression. Patients in group 1 had significantly more often LM or left main equivalent CAD.76% versus 8%(P< 0.001) heart failure 40% versus 4%( P=0.005) and higher in hospital mortality; 24% versus 0%(P= 0.02) than patients in group 11.The troponin levels did not differ significantly between the groups

**6, Correlation of angiographic findings and clinical presentations in unstable angina IRAQI POSTGRADUATE MEDICAL JOURNAL Vol: 10 No 4 , 2011.**

A prospective study of 110 patients of unstable angina at Ibn Albitar Center for cardiac surgery .All the patients underwent cardiac catheterization. Culprit lesion was identified in 80 patients and in 30 pts no identifiable culprit lesion. Complex lesions including complex morphology, intracoronary thrombus, or total occlusion were also analysed and TIMI flow grade was assessed. Patients were classified according to Braunwald class in unstable angina .They compared patients with and with no culprit lesions in regarding Braunwald classification. They sequentially compared the highest Braunwald class 11,C,3 with classes <111 <C<3 respectively regarding the angiographic finding in patients with culprit lesions. Patients with culprit lesions were strongly associated with highest Braunwald class regarding clinical status and intensity of treatment as compared with those with no culprit lesion p value < 0.01. In patients with culprit lesion the angiographic findings were 14 ( 17.5%) intracoronary thrombus, 12( 15%) total occlusion 24(30%) < TIMI 3 flow and 40 (50%) were complex lesions

**7,Unstable angina, Angiographic morphology of the Atherosclerotic lesion and Outcome Hellenic J Cardiology 43:189-194,2002**

Study of 122 patients, men(94),women(28),mean age 61(31-79) yrs who were hospitalized due to unstable angina. Coronary angiogram was performed 3-5 days after admission. The culprit lesion identification based on ECG and angiographic criteria was possible in 114 (93%)pts. The culprit lesions were classified as simple or complex according to Ambrose Modified criteria. Study end points were death, acute MI, CABG, PTCA and angina at one year Simple lesions were present in 42 pts and complex in 72 pts. The outcome of the patients with complex lesions was no different to that of the patients with simple lesions. Revascularisation was done in 74% of the patients [PTCA in 41% and CABG in 33%) Major cardiac events occurred at only during initial hospitalization.

**8,Correlation of TIMI risk score with angiographic severity and extent of coronary artery disease Am J cardiol(2004) 93:813-6 S Garcia ,A peter**

The thrombolysis in myocardial infarction ( TIMI) risk score predicts adverse clinical outcomes in patients with UA/NSTEMI One vessel disease was found in patients with TIMI score 3 to 4 as often in

patients with TIMI score 0-2(OR 1.08,95% CI .However, 1 vessel was found more often in patients with TIMI score 3 to 4 than in pts with 5-7. 2 vessel disease was found more with TIMI 3 to 4 than 0-2 and 5-7 . 3 vessel disease was found more in patients with TIMI score 5-7 than 0-2 and 3-4 . In patients with UA undergoing CAG ,TIMI risk scoring correlated with the extent and severity of CAD

**9, Correlation between the AHCPR (Agency for health care policy and research) risk stratification and angiographic morphology in non ST segment elevation ACS Turk Kardiyol Dern Ars- Arch Turk Soc Cardiol 2011; 39(2):105-113. Ahmed Yildiz ,Ugar Coskun**

Comparison of high risk group with intermediate & low risk group with regard to lesion morphology showed significant higher rates of complex lesions,( 31.9% vs 4.0%, p= 0.001, total occlusion(23.2% vs 0% p= 0.001 ) and intracoronary thrombus (13%vs 2%, p= 0.02) in the high risk group. In univariate analysis, high risk was associated with the presence of complex lesions, total occlusion, intracoronary thrombus and TIMI flow<III. Of these ,only the presence of complex lesions (p= 0.005) and TIMI flow<III(p=0.02) were associated with high risk in multivariate analysis

**10, Clinical profile,Angiographic characteristics and treatment recommendations in patient with coronary artery disease JPMS volume 3,Issue 2.April-june 2013. Ibrahim shah,shahzeb**

On angiographic analysis 18.9% had SVD, 26% had double Vessel disease,and 45% had Triple Vessel Disease , 3% had LM disease and 7% had normal coronaries .The involvement of LAD, LCx and RCA was 42%,26% and 32% respectively. AHA type A,B and C lesions were 37% ,51% and 12%.Other characteristics of lesions include ostial stenosis in 4.7% ,bifurcation lesion in 21%,calcification (7.6%) CTO 6%. PCI and CABG was advised in 66% and 18.9% respectively.

## RESULTS

Table – 1

Age Distribution

| Age in years | No.of cases | Percentage |
|---|---|---|
| 25 – 35 | 5 | 5 % |
| 36 – 45 | 15 | 15% |
| 46 – 60 | 56 | 56% |
| 61 – 75 | 24 | 24% |
| Total | 100 | 100% |

Chi square value - 43.39

'p' value - < 0.001 Significant (46-60 years group)

The age distribution of the patients who underwent the study are plotted in table -1. Patients within the age group of 46-60 years formed 56% of cases with significant lesion in 54 patients. Followed by age group 61-75 yrs, 36-45 yrs and least with 25-35 years.

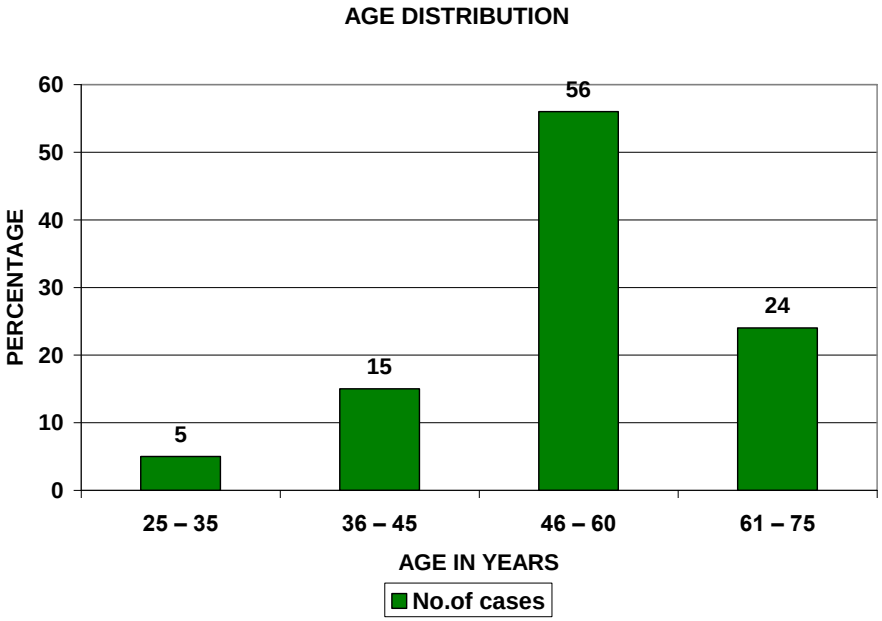

AGE DISTRIBUTION
60
50
40
30
20
10
0
PERCENTAGE
5
15
56
24
25 – 35
36 – 45
46 – 60
61 – 75
AGE IN YEARS
No.of cases

Table - 2

Sex Distribution

| | | CAG | | | |
|---|---|---|---|---|---|
| Sex | No.of cases | LMCA | SVD | DVD | TVD |
| Male | 70 | 10 | 19 | 17 | 20 |
| Female | 30 | 4 | 8 | 7 | 6 |
| Total | 100 | 14 | 27 | 24 | 26 |

Chi square value - 10.061

'p' value - 0.002 Significant (Male)

The sex distribution was plotted in table 2. Male outnumber females in this study. 70 out of 100 cases are males. 66 patients out of 70 patients had significant lesions whereas only 15 patients of the total of 30 patients in female population had significant lesion.

## SEX DISTRIBUTION

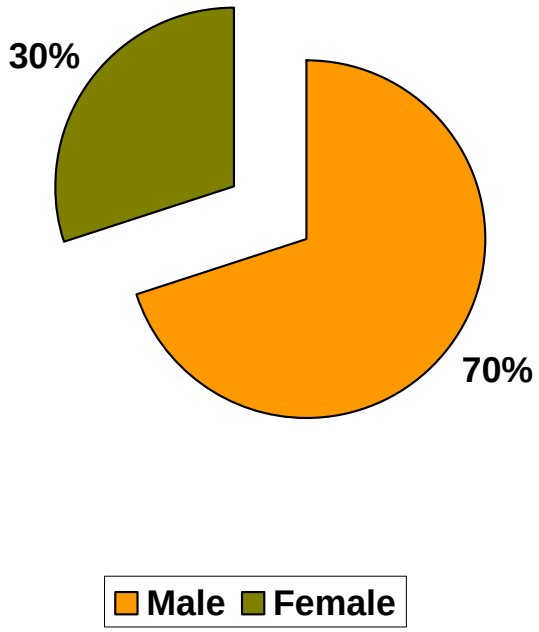


Table – 3

Complaints

| Complaints | No.of cases | Percentage |
|---|---|---|
| Chest pain | 99 | 99 % |
| Dyspnea | 6 | 6% |
| Syncope | 9 | 9% |
| Palpitation | 8 | 8% |

Almost all patients presented with H/o chest pain. Other symptoms like dyspnoea, syncope, palpitation was present in less than 10% of patients.

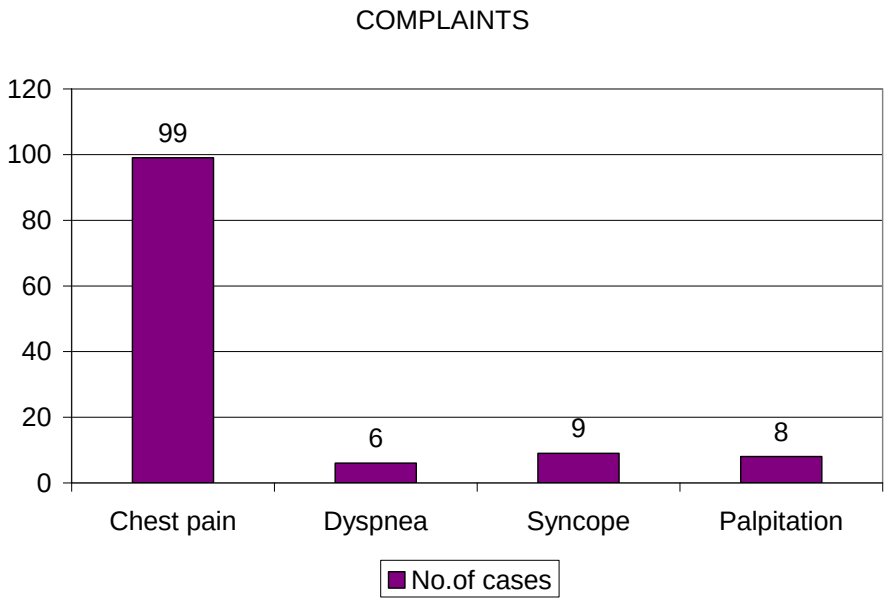

COMPLAINTS
120
100
80
60
40
20
0
99
6
9
8
Chest pain
Dyspnea
Syncope
Palpitation
No.of cases

Table – 4

Risk Factors

| Risk factors | No.of cases | **CAG** | | | | |
|---|---|---|---|---|---|---|
| | | LMCA | SV | 2V | 3V | Normal |
| Smoking | 51 | 6 | 13 | 13 | 15 | 10 |
| DM | 33 | 8 | 5 | 11 | 12 | 5 |
| HT | 26 | 8 | 2 | 9 | 9 | 6 |
| FH | 3 | 0 | 1 | 2 | 0 | 0 |
| Hyperlipidemia | 33 | 8 | 5 | 11 | 12 | 5 |

Smoking, diabetes, hyperlipidemia, hypertension formed the major risk factors in this study. Majority of the patients are smokers and diabetes. 3% of patients had a family history of heart diseases.

RISK FACTORS

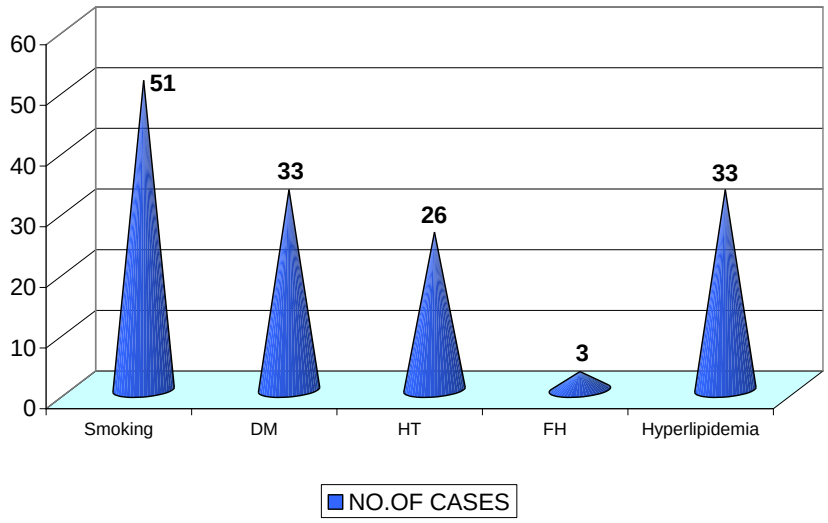


Table – 5

Braunwald Class

| | | CAG | | | |
|---|---|---|---|---|---|
| Braunwald Class | No.of cases | LMCA | SVD | DVD | TVD |
| Class I | 23 | 2 | 4 | 2 | 2 |
| Class II | 46 | 1 | 19 | 13 | 6 |
| Class III | 31 | 11 | 4 | 9 | 18 |
| Total | 100 | 14 | 27 | 24 | 26 |

46 patients presented with class II angina and significant lesion was seen in 40 patients. 31 patients presented with class III symptoms for whom there were significant 3VD and LMCA diseases.

As Braunwald class increased the severity of lesion increased.

Class III vs LMCA

Chi square value - 12.46

P value - 0.002 significant

Class III vs Multivessel disease

Chi square value - 6.23

P value - 0.044 significant

BRAUNWALD CLASS VS SIGNIFICANT CAD

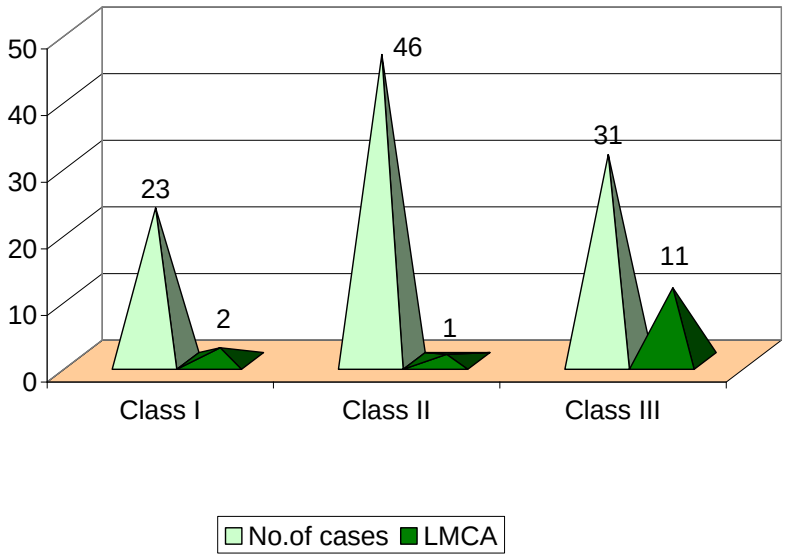


Table – 6

TIMI Score

| TIMI Score | No.of cases | CAG | | | |
|---|---|---|---|---|---|
| | | LMCA | SVD | DVD | TVD |
| 1 | 14 | 0 | 1 | 2 | 0 |
| 2 | 25 | 1 | 10 | 1 | 4 |
| 3 | 32 | 3 | 12 | 12 | 7 |
| 4 | 16 | 3 | 6 | 4 | 5 |
| 5 | 13 | 7 | 2 | 2 | 9 |
| 6 & 7 | 0 | 0 | 0 | 0 | 0 |
| Total | 100 | 14 | 31 | 21 | 25 |

TIMI vs LMCA

Chi square value - 13.92

P value - 0.008 significant

32 patients presented with TIMI-3 score for whom 31 patients had TVD 3 had LMCA diseases. 16 patients presented with TIMI-4 score for whom 13 patients had significant CAD 3 patient 14 patients who presented with TIMI score 1 had lesions in 4 patients.

TIMI SCORE VS CAD

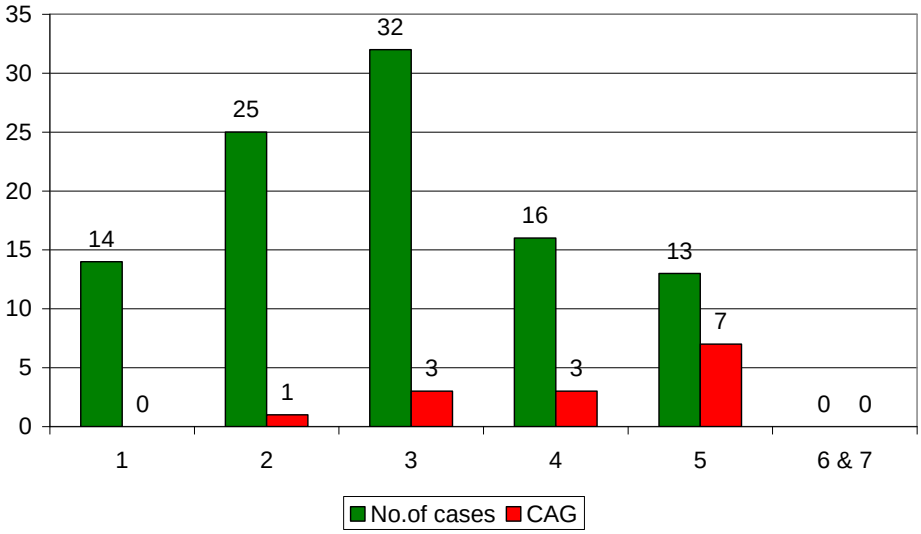


Table – 7

ECG

| ECG | No.of cases | CAG | | | |
|---|---|---|---|---|---|
| | | LMCA | SVD | DVD | TVD |
| ST –T | 34 | 14 | 5 | 10 | 19 |
| AVR | 18 | 14 | 2 | 2 | 13 |
| Normal | 48 | 0 | 0 | 0 | 0 |

Chi square value - 36.93

P value - <0.001 significant

Significant ST-T changes was seen in 34% cases

ST elevations in AVR seen in 18 patients. Normal ECG was seen in 48% patients.

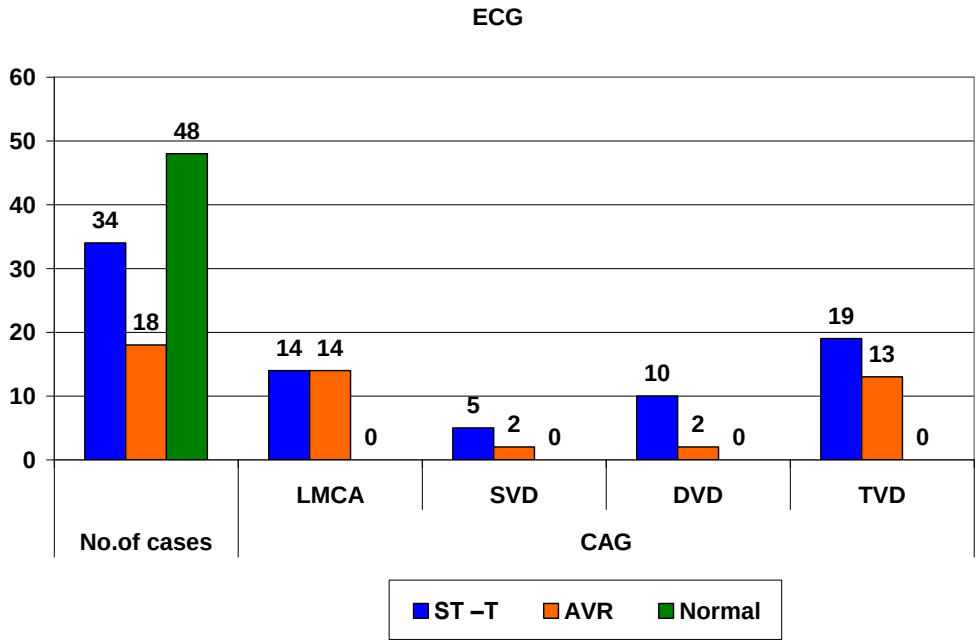

ECG
60
50
40
30
20
10
0
34
18
48
14
14
0
5
2
0
10
2
0
19
13
0
No.of cases
LMCA
SVD
DVD
TVD
CAG
ST –T
AVR
Normal

Table – 8

ECHO

| ECHO | No.of cases | CAG | | | |
|---|---|---|---|---|---|
| | | LMCA | SVD | DVD | TVD |
| LV dysfunction | 26 | 6 | 7 | 9 | 9 |
| Normal | 74 | 8 | 20 | 15 | 17 |

LV Dysfunction was seen in 26 patients, who showed LMCA lesion in 6 patients, 3VD in 25 patients.

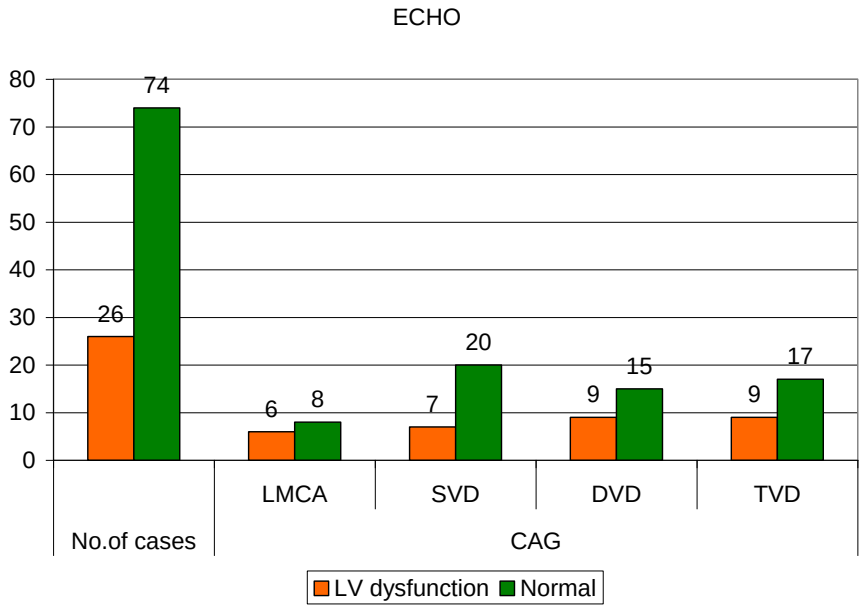
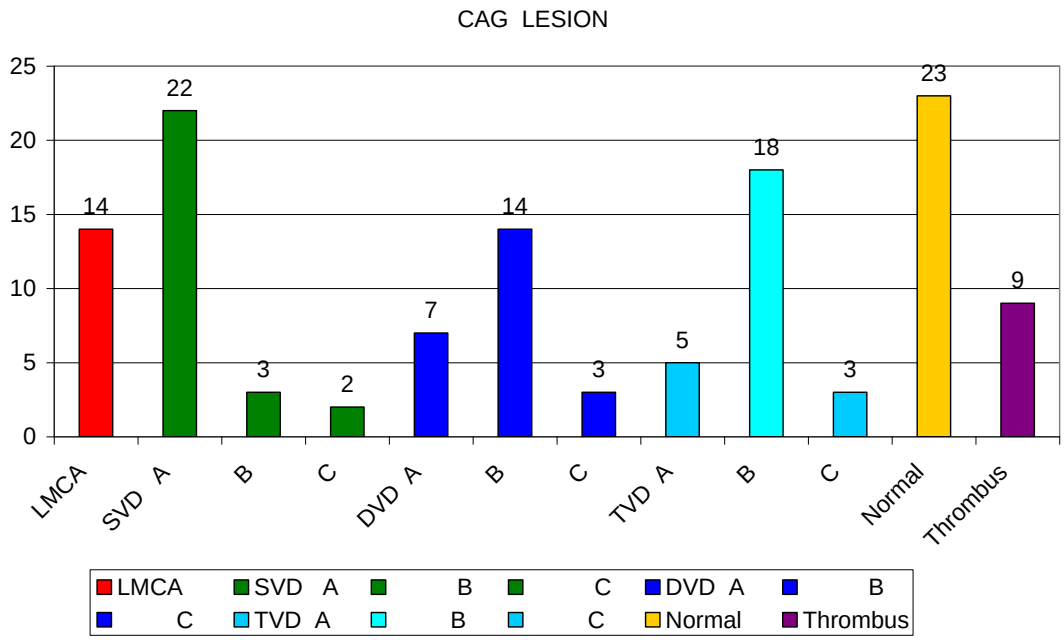

ECHO
80
70
60
50
40
30
20
10
0
74
26
6
8
7
20
9
15
9
17
LMCA
SVD
DVD
TVD
No.of cases
CAG
LV dysfunction
Normal

Table – 9

CAG

| CAG | Type | No.of cases |
|---|---|---|
| LMCA | | 14 |
| SVD (27) | A | 22 |
| | B | 3 |
| | C | 2 |
| 2VD (24) | A | 7 |
| | B | 14 |
| | C | 3 |
| 3VD (26) | A | 5 |
| | B | 18 |
| | C | 3 |
| Normal | | 23 |
| Thrombus | | 9 |
| Total Occlusion (TIMI 0 flow) | | 3 |

LMCA was seen in 14 patients in this study. 27 patients had single vessel disease, 24 patients had 2VD, 26 patients had 3VD, 23 patients had normal / insignificant coronary lesions. In single vessel disease most of the lesions are type A, In 2VD and 3VD mostly the lesions are type B.


CAG LESION
25
20
15
10
5
0
14
22
3
2
7
14
3
5
18
3
23
9
LMCA
SVD A
B
C
DVD A
B
C
TVD A
B
C
Normal
Thrombus
LMCA
SVD A
B
C
DVD A
B
C
TVD A
B
C
Normal
Thrombus

# DISCUSSION

Each year, roughly one million patients in the united states are hospitalized for unstable angina or non-ST elevation myocardial infarction .The incidence of NSTE-ACS both absolute and relative to STEMI, is increasing probably as a result of demographic changes in the population, including progressively increasing numbers of older patients and higher rates of diabetes and other novel risk factors.

The study showed 56% of patients fall into the age group 46– 60 yrs, 24% of patients in age group 61-75 yrs, 15% of patients in age group of 36-45 followed by 5 % of patients in age group of 25- 35 yrs Of the patient who were diagnosed as unstable angina 70 % were males, 30% females . This correlated with a Iraqi study by Kasim Abbas Ismail et al[42] who studied 110 patients which showed a prevalence of 72.5 % and 27.5%. In some western studies females represented 30% -45% of cases. Chi square test value is 10.061 and p value is 0.002 (Significant).

This study showed smoking 51 pts ,followed by diabetes 33 pts, hyperlidemia 33 pts, and hypertension 26 pts as the major risk factor in unstable angina.

Majority of patients in this study presented with Braunwald class II angina (46 pts) class III angina ( 31 pts) class I angina (23 patients). Out of the 46 pts who presented with class II angina 39 pts had two &

three vessel disease. Out of the 31 pts who presented with class III angina all had triple vessel disease & eleven patient had Left main disease. More severe the Braunwald class, patients had two and three vessel disease and left main coronary artery disease. The chi square test value 12.466 and p value is 0.002 significant.

This study correlated well with a study done by Calton R and Jaison TM et al[12] in 1998 ( Indian heart Journal) in a study of 100 pts with unstable angina 17 pts (class I), 58 pts (class II), 25 pts (class III) . Single vessel disease is seen in Braunwald class I pts , compared to class II & III pts

The majority of patients presented with TIMI risk 2-4/7 . 32 patients presented with TIMI 3/7, out of whom 3 had LMCA disease, 31 had 2VD and 3VD. 16 pts presented with 4/7. of them 3 had LMCA, and 15 had 2VD and 3VD. 13 pts presented with TIMI 5/7, out of whom 7 had LMCA disease and 13 had TVD

From this study it was concluded that higher the TIMI risk scoring more the prevalence of TVD and Left main disease Chi square test 13.924 , P value 0.008 ( significant)

This study correlated reasonably well with the study done by S.Garcia,A. Peter et al[43] in AJC 1 vessel was found more in patients with TIMI score 3 to 4 than in pts with 5-7. 2 vessel was found more

with TIMI 3 to 4 than 0-2 and 5-7 . 3 vessel disease was found more in pts with TIMI score 5-7 than 0-2 and 3-4

In our study the ECG showed significant ST- T changes in 52 % of the patients with aVR elevation in 18 pts . Out of the 18 pts 14 pts had LMCA disease and another 4 had TVD. 48% had insignificant ECG changes .

This study correlated well with a study done by calton R and Jaison TM et al[12] in 1998 ( Indian heart Journal) in a study of 100 pts with unstable angina who underwent coronary angiogram showed 54% of the patients had ST-T changes, 46% had normal or insignificant coronary lesions.

Patients with ECG changes showed significant lesions (2VD, 3VD, LM and ACC class B/C lesions) compared with normal ECG. Weber T, Maurer E et al[11] in a study of 30 pts who risk stratified according to severity of ECG changes who subsequently underwent coronary angiogram showed the more severe the ECG changes, the more extensive the coronary lesions

Out of the 100 Patients 26 pts presented with RWMA & LV dysfunction on ECHO screening 25 had significant two and three vessel disease &LMCA disease

Out of the 100 pts who underwent coronary angiogram 27 pts had Single Vessel Disease . 24 pts had two Vessel disease .26% had three vessel disease. 14 patients had Left Main Coronary artery disease . 23 patients had normal or insignificant coronary artery lesions.

The extent of epicardial CAD among patients with UA /NSTEMI randomized to the invasive arm of the TACTICS-TIMI 18 trial who systematically underwent was as follows 34% had triple vessel disease, 28% had two vessel disease, 26 had single vessel disease, 13 had no coronary stenosis, 10 % had LM stenosis. This correlated reasonably well with our study. Registries of unselected UA pts have reported same findings

Ibrahim shah, shahseb et al[44] in a study On angiographic analysis in unstable angina showed 18.9% had SVD,26 % had double Vessel disease, and 45% had Triple Vessel Disease, 3% had LM disease and 7% had normal coronaries.

**Study Limitations**

Like most of the studies application of coronary angiogram in lesion assessment has its own limitations.

Though coronary angiogram is considered the gold standard tool for the assessment of coronary lesions . It is a luminogram . The plaque volume & characteristics cannot be assessed by coronary angiogram.

Multiple planes has to be assessed before lesion assessment in case of eccentric lesions.

In this study only qualitative morphological lesions were assessed . The QCA (Quantitative Coronary Angiography) method increases the lesion assessment more accurately. IVUS (intravascular ultrasound imaging) is the best modality to assess the lesion severity in intermediate lesions (50-70) to decide on further management. FFR (fractional flow reserve) gives more physiological information regarding the lesion severity. The absence of both IVUS and FFR are major drawback in this study.

# CONCLUSION

This is a single center observational cross sectional study where 100 patients who were admitted as unstable angina over a period of 6 months at our hospital who subsequently underwent coronary angiogram, to study the correlation of the clinical presentation and coronary angiographic profile. After clinical evaluation, braunwald clinical classification, TIMI risk score, ECG, biomarkers, ECHO the patients were risk stratified into low, intermediate and high risk. The clinical presentation and angiographic profile of the patients are correlated. The following conclusions are derived.

Unstable angina commonly affects the age group 45-60yrs in both sexes. 30% of patients in our study was women

Women have normal coronaries compared to men in patients with unstable angina, (30% vs 20%) which suggests a different patho physiological mechanism for their symptoms which leads to difficulty in making a firm diagnosis of UA.

Smoking, diabetes, Hyperlipidemia, Hypertension are major risk factors for unstable angina in this study

Braunwald class III angina (Rest angina) predicted severity of lesion ( left main & triple vessel disease) in our study

Patients who had High TIMI risk scoring had more severe coronary lesions compared to low TIMI risk score which helps in risk stratification and early intervention

Significant ST-T changes in ECG predicted more extensive disease which helps in decision making regarding treatment strategy (conservative vs invasive) aVR ST elevation in background of unstable angina predicts left main disease &Triple vessel disease in our study which helps risk stratification and early intervention

ECHO evidence of LV dysfunction predicted Triple vessel disease /LM disease.

Out of the 100 pts who underwent coronary angiogram in our study 27 pts had Single Vessel disease ( type A lesions predominantly) 24 pts had two Vessel disease .( type B lesions predominantly) 26% had three vessel disease. (type B lesions predominantly) 14 patients had Left Main Coronary artery disease.

23 patients had normal or insignificant coronary artery lesions. 9 patients had thrombus containing lesion who had rest angina, out of whom 6 patients had SVD and 3 patients had multivessel disease.

3 patients had total occlusion with TIMI ‘0’ flow.

# RECOMMENDATIONS

The results of this study gives us an idea about the clinical presentation of the patients with unstable angina correlated with coronary angiographic findings

Higher Braunwald class (rest angina), higher TIMI risk score, Significant ECG changes (ST-T ) changes & aVR ST elevation ,features of LV dysfunction signifies significant and extensive lesions which helps in risk stratification & early intervention. These clinical and other parameters helps us to predict in the bedside the severity of the lesion and the culprit vessel involved so that early invasive intervention can be planned.

When practicing in an remote area without cathlab facilities ,based on these clinical parameters it helps us to identify high risk patient who will benefit the most from early intervention ,by timely referral or by aggressive use of antithrombotics.

Also in the precath evaluation we can be mentally prepared about the anatomy of the culprit vessel involved so that utmost precaution can be carried out during the procedure to prevent complications.

# BIBLIOGRAPHY


1, Canon C.P et al The ECG predicts one year outcome of pts with unstable angina, Results of TIMI III registry JACC 30:133,1997

2, Gugliano RP, Braunwald E The year in non ST segment elevation ACS JACC 54:1544,2009.

3, Braunwald E Unstable angina : An etiological approach to management Circulation 98 :2219,1918

4, Ambrose JA, Winters SI,Stern A,et al. Angiographic morphology and the pathogenesis of unstable angina pectoris JACC 1985;5:609-616

5, Mueller C,Neumann FI,et al .Prognostic value of the admission ECG in pts with UA treated with very early revascularization Am.J Med 2004;117:145-150

6, Fuchs S,Stabile E,et al .Intravascular ultrasound finding in patients with acute coronary syndromes with and without elevated troponin I level Am J Cardiology 2002 ;89: 1111-1113.

7, Cohen M,Demers C et al et al A comparison of LMWH with Unfractionated heparin for unstable coronary artery disease NEJM 1997;337:447-452

8, Boersma E,Pieper KS et al .Predictors of outcome in patients ACS without ST elevation. The PURSUIT investigators, Circulation 2000;101:2557-2567

9, Angiographic progression in patients with angina and normal or near normal coronary angiograms who are restudied due to unstable symptoms. Cox ID, Schwartzman RA, Atienza F, Brown SJ, Kaski JC. Eur Heart J. 1998 Jul;19(7):1027-33.

10. Angiographic occurrence and clinical correlates of intraluminal coronary artery thrombus: role of unstable angina. Bresnahan DR, Davis JL, Holmes DR Jr, Smith HC. J Am Coll Cardiol. 1985 Aug;6(2):285-9.

11. Clinical presentations and coronary angiographic results in unstable angina Weber T, Kirchgatterer A, Auer J, Wimmer L, Lang G, Mayr H, Maurer E, Punzengruber C, Eber B.Acta Med Austriaca. 1999;26(1):12-6. German.

12. Corelation of Braunwalds clinical classification of unstable angina with angiographic extent of disease,lesion morphology and intraluminal thrombus Calton R, Satija T, Dhanoa J, Jaison TM, David T. Indian Heart J. 1998 May-Jun;50(3):300-6.

13. Corelation of coronary angiographic morphology with clinical presentation in unstable angina Liu X, Cui Z, Hu D, Li T. Zhonghua Nei Ke Za Zhi. 2001 May;40(5):306-9. Chinese.

14. Corelation of clinical and angiographic morphology in unstable angina Ben-Hamda K, Gharbi M, Hendiri T, Addad F, Denguir H, Mlika A, Abbes F, Zeghidi B, Derdabi M, Betbout F, Gamra H, Maatouk F,

Dridi Z, Ben-Farhat M.Tunis Med. 2004 Jan;82 Suppl 1:164-75. French.

15. Angiographic and Scintigraphic correlates of clinical presentation in unstable angina Emre A, Ersek B, Gürsürer M, Aksoy M, Siber T, Engin O, Yeşilçimen K. Clin Cardiol. 2000 Jul;23(7):495-500.

16. Clinical risk stratification correlates with angiographic extent of coronary artery disease in unstable angina Mathew V, Farkouh M, Grill DE, Urban LH, Cusma JT, Reeder GS, Holmes DR Jr, Gersh BJ. J Am Coll Cardiol. 2001 Jun 15;37(8):2053-8.

17. Angiographic occurrence and clinical correlates of intraluminal coronary artery thrombus: role of unstable angina Bresnahan DR, Davis JL, Holmes DR Jr, Smith HC.
J Am Coll Cardiol. 1985 Aug;6(2):285-9.

18. Angiographic progression in patients with angina and normal or near normal coronary angiograms who are restudied due to unstable symptoms Cox ID, Schwartzman RA, Atienza F, Brown SJ, Kaski JC. Eur Heart J. 1998 Jul;19(7):1027-33.

19. The Angiographic spectrum of Coronary Artery Disease Reeder GS, Smith HC, Elveback LR, Mock MB.Cardiovasc Clin. 1985;15(1):17-31.

20. Relation between serum troponon I values and severity of ECG, clinical , quantitative angiogram in Unstable angina Janorkar S,

Koning R, Eltchaninoff H, Andres H, Lavoinne A, Cribier A. Indian Heart J. 1999 Jan-Feb;51(1):31-4.

21, Granger CB,Goldberg RJ,Dabbous O et al .Predictors of hospital mortality in the global registry of acute coronary events .Arch Intern Med 2003;163:2345-53.

22, Savonitto S,Ardissino d,Granger CB ,et al .Prognostic value of the admission ECG in ACS JAMA .I999:281:707-13.

23, Lloyd Jones D et al . Heart disease and stroke statistics- 2010 update : a report from the American Heart Association , Circulation 2010; 121 : e 46-215.

24, Greenland P,Knoll MD et al .Major risk factors as antecedents of fatal and nonfatal coronary artery disease events .JAMA .2003;290:891-7.

23, de Zwaan,Janssen JH et al. Angiographic and clinical characteristics of patients with unstable angina showing an ECG pattern indicating critical narrowing of the proximal LAD coronary artery .Am.Heart J.1989;117:657-65

26, Jaffe AS et al .Biomarkers in the acute cardiac disease ,the present and the future J Am Coll Cardiology 2006;48:1-11

27, Westerhout CM et al . Short and long term risk stratification in acute coronary syndromes : the added value of quantitative ST segment depression and multiple biomarkers J Am Coll Cardiol.2006;48:939-47

28, Antmann EM,Cohen et al .The TIMI risk score for unstable angina /non-ST elevation MI; a method for prognostication and therapeutic decision making JAMA 2000;284:835-42

29, Granger CB ,Goldberg RJ et al ,Predictors of hospital mortality in the global registry of acute coronary events ,Arch Intern Med 2003; 161: 2345-53

30, Knot UN et al .Prognostic importance of physical examination for heart failure in non ST elevation acute coronary syndromes: the enduring value of clinical examination

31, Ohman EM et al Cardiac troponin T levels for risk stratification in myocardial ischemia .GUSTO II A investigators .N Eng J Medicine 1996;335:1333-41

32, Wright RS ,Anderson JL et al 2011 ACCF/AHA Focussed update of the guidelines of the management of unstable angina Circulation 2011

33, Buffon A,Biassucci LM et al Widespread coronary inflammation in unstable angina .N .Eng Journal of medicine 2002;347:5-12

34, Goldstein JA et al .Multiple complex coronary plaques in patients with acute myocardial infarction .N Engl J Med 2000;343:915-22

35, Alderman EL et al Five year angiographic followup of factors associated with progression of CAD in the CASS study J AM Coll Cardiolb1993 ; 22: 1141-54

36, Vasan RS et al .Relative importance of borderline and elevated levels of CAD risk factors Ann Intern Med 2005 : 142: 393-402

37, Smith SC et al ACC /aha 2005 guideline update for PCI : a report of the ACC guidelines for PCI J AM Coll Cardiol 2006 ;47:e l-121

38, Ohman EM et al .Cardiac troponin T levels for risk stratification in acute myocardial ischemia GUSTO II A Investigators .N Eng J Medicine 1996;335:1333-41

39, Eagle KA ,Lim MJ et al .A validated prediction model for all forms of acute coronary syndrome: estimating the risk of 6-month post discharge in an international registry JAMA 2004;291:2727-33

40, Spacek R,Widimsky P et al Value of first day angiography/angioplasty in evolving NSTEMI :an open multicenter randomized trial. The VINO study Eur Heart J.2002:23:230-8

41, Naghavi M, Libby P et al From vulnerable plaque to vulnerable plaque , circulation 2003:108:1772-8

# PROFORMA

Name
Age
Gender
Education
Marital status
Occupation
Address

Date of admission
Date of discharge/death
IP no.
Ward/Unit

Duration of hospital stay

**SYMPTOM PROFILE**

**1.** Chest pain – typical Atypical

2. Dyspnea - NYHA class

3. Palpitation

4. Giddiness

5. Syncope

6. Angina equivalents

7. BRAUNWALDS CLASS 1 A B C , 11 A B C , 111 A B C

**TIMI RISK SCORE**

Age > 65

> 3 CAD risk factors

Prior Coronary stenosis > 50%

Aspirin in last 7 days

2 anginal episodes < 24 hrs

ST segment deviation

Elevated cardiac markers

**RISK FACTORS**

1. Diabetes
2. Hypertension
3. Smoker
4. Alcoholism
5. PAD/other vascular diseases
6. Drug abuse
7. Family history of CAD
8. Hypercoagulable states
9. Connective tissue disorders
10. Prior MI /PCI/CABG

PHYSICAL EXAM

BP

PR

CVS

RS

CNS

Killips Class

INVESTIGATIONS

1. Blood Sugar
2. Blood Urea
3. Serum Creatinine
4. Lipid profile:
5. ECG Normal ST changes T wave changes
6. Echocardiogram Normal WMSI Arterial territory LAD, LCx, RCA
7. Coronary Angiogram : Normal, LMCA , 1VD , 2VD,3VD Type A, Type B , Type C

OUTCOME

1. Identifying the correlation of clinical parameters to angiographic profile
2. Identification of High risk predictors

ANALYSIS

1. Age
2. Gender
3. Risk factors involved
4. Braunwald clinical classification
5. Clinical risk stratification
6. ECG, ECHO parameters
7. CAG – profile

## ABBREVIATION

| | | |
|---|---|---|
| ACS | - | Acute Coronary Syndrome |
| CABG | - | Coronary Artery bypass graft |
| CSA | - | chronic stable angina |
| NSTEMI | - | Non ST elevation MI |
| STEMI | - | ST elevation MI |
| aVR | - | augmented precordial right lead |
| ED | - | Emergency department |
| IVUS | - | Intravascular Ultrasound |
| LMCA | - | Left Main Coronary Artery |
| DVD | - | Double Vessel Disease |
| SVD | - | Single Vessel Disease |
| TVD | - | Triple Vessel Disease |
| TIMI | - | Thrombolysis in myocardial infarction |
| LVEF | - | Left Ventricular Ejection Fraction |
| Trop I | - | Troponin I fraction |
| UA | - | Unstable angina |

# MASTER CHART

| S L NO | NAME | AGE | SEX | COMPLAINTS | | | | RISK FACTORS | | | | | BRAUNWALD CLASS | | | TIMI SCORE | | | | | | | ECG | | ECHO | BIO MARKERS | CAG | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | C P | DYPSNEA | SYNCOPE | PALPITATION | SMOKING | DM | HTN | F H | HYPER LIPIDEMIA | CLASS -1 | CLASS -2 | CLASS -3 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | ST-T | AVR | LV DYSFUNCTION | TROPONINS | LMCA | SVD | | | 2VD | | | 3VD | | | NORMAL |
| | | | | | | | | | | | | | | | | | | | | | | | | | | | A | B | C | A | B | C | A | B | C | |
| 1 | valli | 35 | F | y | | | | | | | | | Y | | | | Y | | | | | | | | | | | | | | | | | | | | | N |
| 2 | Panchavamum | 49 | F | y | y | | | | | y | | | | | y | | | | | Y | | | Y | Y | | Y | Yes | | | | | | | | | yes | | |
| 3 | Pitchai | 65 | M | y | | | | y | y | | | y | | Y | | | Y | | | | | | | | | | | | | | | | | | | | N |
| 4 | Dhesingu | 59 | M | y | | | | y | | | | | Y | | | Y | | | | | | | | | | | | | | | | | | | | | N |
| 5 | Ramalakshmi | 38 | F | y | | | | | | | | | | Y | | | Y | | | | | | | | | | | Yes | | | | | | | | | | |
| 6 | Sarasu | 45 | F | | | | y | | | | | | Y | | | Y | | | | | | | | | | | | | | | | | | | | | N |
| 7 | Malliga | 47 | F | y | | | | | | | | | | Y | | | | Y | | | | | | | | | | | Yes | | | | | | | | | |
| 8 | Thirunavukarasu | 48 | M | y | | | | | | | | | | Y | | | Y | | | | | | | | | | | | | | | | | | | | N |
| 9 | Arokiaraj | 65 | M | y | | y | | y | y | y | | y | | Y | | | | | | Y | | | | Y | | | | | | | | | | | | | yes | | |
| 10 | Muthu | 46 | M | y | | | | y | | | | | Y | | | | Y | | | | | | | | | | | | | | | | | | | | N |
| 11 | Pandi | 63 | M | y | | | | y | y | | | y | Y | | | | | | | Y | | | | | | | | | | | | | | | | | N |
| 12 | Boominathan | 54 | M | y | | | | y | | | | | | | Y | | | | | Y | | | | Y | | Y | | | | | Yes[T] | | | | | | | |
| 13 | John peter | 50 | M | y | | | | | | | | | | Y | | | | Y | | | | | | | | | | Yes | | | | | | | | | | |
| 14 | Baskar | 56 | M | y | | | | y | | | | | | Y | | | | Y | | | | | | | | | | | | | | | Yes | | | | | |
| 15 | Sanjeevi | 60 | M | y | | | | | y | | | y | | | y | | | | Y | | | | Y | Y | Y | Y | yes | | | | | | | | | Yes | | |
| 16 | Vasantha | 62 | M | y | | | | | y | | | y | | Y | | | | | Y | | | | | | | | | | Yes | | | | | | | | | |
| 17 | Mathialagan | 53 | M | y | y | | | | | y | | | | | y | | | | | Y | | | Y | Y | | | Yes | | | | | | | | | yes | | |
| 18 | Loganathan | 63 | M | y | | | | | y | | | y | | | Y | | | | | Y | | | Y | | Y | | | | | | | | Yes/TC | | | | | |
| 19 | Jeyaraman | 52 | M | y | | | | | | | | | | Y | | | | | Y | | | | | | | | | | | | | | | | Yes | | | |
| 20 | Raji | 45 | M | y | | | | y | | | | | Y | | | | | Y | | | | | Y | Y | | | yes | Yes | | | | | | | | | | |
| 21 | Saravanakumar | 30 | M | y | | | | y | | | | | | Y | | Y | | | | | | | | | Y | | | | | | | | | | | | | N |
| 22 | Karuppiah | 62 | M | y | | y | | y | y | | | y | | Y | | | | Y | | | | | | | | | | | | | | yes | | | | | | |
| 23 | Sunderrajan | 52 | M | y | y | | | y | | y | | | | | y | | | | | Y | | | Y | Y | Y | | Yes | | | | | | | | | yes | | |
| 24 | Dhanasekharan | 57 | M | y | | | | y | | | | | | | y | | | | Y | | | | | | | | | | | | | | | | Yes | | | |
| 25 | Arumugam | 58 | M | y | | | | y | y | | | y | | | Y | | | Y | | | | | | | Y | | | Yes[T] | | | | | | | | | | |
| 26 | Thomas | 43 | M | y | | | | y | | | | | Y | | | Y | | | | | | | | | | | | | | | | | | | | | N |

| | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 27 | Pitchaiammal | 63 | F | y | y | | | | y | y | | y | | | y | | | | Y | | | | Y | Y | | | Yes | | | | | yes | | | | | |
| 28 | Marimuthu | 38 | M | y | | | | y | | | | | | Y | | | | Y | | | | | | | | | | | Yes | | | | | | | | | |
| 29 | Athilekshmi | 65 | F | y | | y | | | | | | | Y | | | Y | | | | | | | | | | | | | | | | | | | | | | N |
| 30 | Omprakash | 56 | M | y | | | | y | | | | | | Y | | | Y | | | | | | | | | | | | Yes | | | | | | | | | |
| 31 | Vishalakshi | 55 | F | y | | | | | y | | | y | Y | | | | Y | | | | | | | | | | | | | | | | | | | | | N |
| 32 | Jehangirkhan | 54 | M | y | | | | y | | | | | | Y | | | Y | | | | | | | | | Y | | | Yes | | | | | | | | | |
| 33 | Padmamani | 53 | M | y | | | | y | | | | | Y | | | Y | | | | | | | | | | | | | | | | | | | | | | N |
| 34 | Subbulakshmi | 43 | F | y | | | | | | y | | | Y | | | Y | | | | | | | | | | | | | | | | | | | | | | N |
| 35 | Murugan | 40 | M | y | | | | y | | | | | | Y | | | Y | | | | | | | | | | | | Yes | | | | | | | | | |
| 36 | Anantham | 48 | F | y | | | y | | | | | | | Y | | | | Y | | | | | | | | | | | | | Yes | | | | | | | |
| 37 | Puspham | 65 | F | y | | y | y | | y | y | | y | | | Y | | | Y | | | | | Y | | Y | Y | | | | | | Yes | | | | | | |
| 38 | Nadarajan | 66 | M | y | | y | | y | | y | | | | | Y | | | Y | | | | | Y | | | | | | | | | Yes | | | | | | |
| 39 | Murugeswari | 45 | F | y | | | | | | | | | | Y | | | | Y | | | | | | | | | | | Yes | | | | | | | | | |
| 40 | Kuttiyammal | 54 | F | y | y | | | | y | | | y | | | y | | | | Y | | | | Y | Y | Y | | Yes | | | | | | | | Yes | | |
| 41 | Vellaisamy | 55 | M | y | | | | y | | y | | | | Y | | Y | | | | | | | | | | | | | | | | | | | | | N |
| 42 | Agalya | 60 | F | y | | | | | y | | | y | | | y | | | Y | | | | | Y | | | | | | | | | | | | | Yes | | |
| 43 | Murugan | 57 | M | y | | | | | | | | | Y | | | | | Y | | | | | | | | | | | Yes | | | | | | | | | |
| 44 | Arunachalam | 55 | M | y | | | | y | y | | | y | Y | | | | | | | Y | | | Y | Y | | Y | Yes | | | | | | | | Yes | | |
| 45 | Nagammal | 60 | F | y | | | y | | y | y | | y | | Y | | | Y | | | | | | | | | | | Yes | | | | | | | | | |
| 46 | Jeyalekshmi | 65 | F | y | | | | | y | | | y | | Y | | | | Y | | | | | | | Y | | | | | | | Yes | | | | | | |
| 47 | Sekhar | 50 | M | y | | | | y | | | | | Y | | | | Y | | | | | | | | | | | Yes | | | | | | | | | |
| 48 | Arumugam | 55 | M | y | | | | y | | | | | | Y | | | Y | | | | | | | | | | | Yes | | | | | | | | | |
| 49 | Rani | 35 | F | y | | | | | | | | | | | Y | Y | | | | | | | | | | Y | | | Yes[T] | | | | | | | | | |
| 50 | Jarina begum | 56 | F | y | | | y | | y | y | | y | | Y | | | | Y | | | | | Y | | Y | | | | | | | Yes | | | | | | |
| 51 | Krishnan | 49 | M | y | | | | y | | | | | | Y | | | Y | | | | | | | | | | | Yes | | | | | | | | | |
| 52 | Selvam | 48 | M | y | | | | y | | | | | | Y | | | | Y | | | | | | | Y | | | | | | | Yes | | | | | | |
| 53 | Suryaprabha | 60 | F | y | | | | | y | | | y | | | y | | | Y | | | | | Y | Y | | | | | | | | | | | Yes | | |
| 54 | Murugan | 50 | M | y | | | | y | | | | | | | y | | Y | | | | | | Y | Y | Y | | | | | | | | | | Yes | | |
| 55 | Valli | 72 | F | y | | y | | | y | y | | y | | Y | | | | Y | | | | | | | | | | | | | | Yes | | | | | | |
| 56 | Sahul hameed | 63 | M | y | | | | y | y | | | y | Y | | | | | Y | | | | | | | Y | | | | | | | | Yes | | | | | |
| 57 | Chinnasamy | 52 | M | y | | | | y | y | | | y | | | y | | | Y | | | | | | | | | | | | | | | | | Yes | | |
| 58 | Arumugham | 60 | M | y | | | | y | y | | | y | | | y | | | Y | | | | | Y | | Y | | | | | | | | | | Yes | | |
| 59 | Ramar | 51 | M | y | | | | | | | | | | Y | | | Y | | | | | | | | | | | Yes | | | | | | | | | |
| 60 | Sithiga beevi | 61 | F | y | | | y | | | y | | | | Y | | | Y | | | | | | | | | | | | | | | | | | | | | N |
| 61 | Sampath | 50 | M | y | | | | y | | | | | | Y | | | | Y | | | | | | | | | | | | | | | | Yes | | | | |
| 62 | Chandran | 50 | M | y | | | | y | | | | | | | y | | | | | Y | | | Y | Y | | | Yes | | | | | | | | Yes | | |
| 63 | Balakrishnan | 40 | M | y | y | | | y | | y | | | | | y | | | | | Y | | | Y | Y | Y | Y | Yes | | | | | | | | Yes | | |
| 64 | Perumal | 50 | M | y | | | | y | y | | | y | | Y | | | | Y | | | | | | | | | | | | | | yes | | | | | | |
| 65 | Nallasamy | 63 | M | y | | | | | | y | | | | Y | | | Y | | | | | | | | | | | | | | | | | | | | | N |
| 66 | Mydeen pitchai | 39 | M | y | | | | y | | | | | Y | | | Y | | | | | | | | | Y | | | | | | | Yes | | | | | | |

| | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 67 | Asokan | 63 | M | y | | y | | | y | y | | y | | Y | | | | Y | | | | | | | | | | | | | | | | | | | | N |
| 68 | Pandiammal | 40 | F | y | | | | | | | | | Y | | | | Y | | | | | | | | | | | | | | | | | | | | | N |
| 69 | Kalidhas | 46 | M | y | | | | y | | | | | | | y | | Y | | | | | | | | Y | | | | | | | | | | Yes | | | |
| 70 | Mahadevan | 61 | m | y | | | | | y | y | | y | | Y | | | | Y | | | | | Y | Y | Y | | yes | | | | | | | | Yes | | | |
| 71 | Neelamegakan | 28 | M | y | | | | | | | y | | | | Y | Y | | | | | | | | | | | | | | | Yes | | | | | | | |
| 72 | Nalliyan | 65 | M | y | | | | y | y | y | | y | | | y | | Y | | | | | | Y | Y | | | yes | | | | | | | | Yes | | | |
| 73 | Rajaram | 35 | M | y | | | | y | | | y | | | Y | | | | Y | | | | | | | Y | | | Yes[T] | | | | | | | | | | |
| 74 | Rajendran | 59 | M | y | | | | y | | | | | | | Y | | | | Y | | | | Y | | | | | | | | | Yes[T] | | | | | | |
| 75 | Murugesan | 52 | M | y | | | | | | | | | | Y | | | | | Y | | | | | | Y | | | Yes[T] | | | | | | | | | | |
| 76 | Rethinam | 58 | M | y | | | | y | | y | | | | | Y | | | | Y | | | | | | | | | | | | | Yes[T] | | | | | | |
| 77 | Raman | 53 | M | y | | | | | y | | | y | | Y | | | | | Y | | | | Y | | | | | Yes[T] | | | | | | | | | | |
| 78 | Vellaisamy | 65 | M | y | | | | y | y | y | | y | Y | | | | Y | | | | | | | | | | | | | | | | | | | yes | | | |
| 79 | Nagammal | 72 | F | y | | | y | | y | y | | y | | Y | | | Y | | | | | | | | | | | | | | Yes | | | | | | | |
| 80 | Seenivasan | 42 | M | y | | | | y | | | | | Y | | | | Y | | | | | | | | | | | | | | | | | | | | | N |
| 81 | Malayayee | 58 | F | y | | | | | | | | | | Y | | | | | | Y | | | | | | | | | | | | | | | Yes | | | |
| 82 | Arumugham | 65 | M | y | | | | | y | y | | y | | | y | | | Y | | | | | | Y | Y | Y | | yes | | | | | | | | Yes | | | |
| 83 | Saroja | 50 | F | y | | | | | | | | | | Y | | | | | | Y | | | | | | | | | | | | | | | Yes | | | |
| 84 | Chinnaya | 50 | M | y | | | | y | | | | | | | y | | | | Y | | | | Y | | | | | | | | | | | | Yes | | | |
| 85 | Kala | 44 | F | y | | | | | | | | | | Y | | | | Y | | | | | | | | | | | | | | | Yes | | | | | | |
| 86 | Sekar | 48 | M | y | | | | y | | | | | Y | | | | Y | | | | | | | | | | | | | | | | | | | | | N |
| 87 | Selvam | 49 | M | y | | | | y | | | | | | Y | | | | | | Y | | | Y | | | | | | | | | | | | yes | | | |
| 88 | Chandra | 55 | F | y | | | | | | y | | | Y | | | | | | | Y | | | | | Y | | | Yes | | | | | | | | | | |
| 89 | Karrupu | 42 | M | y | | | | y | | | | | | | Y | | | | Y | | | | Y | Y | | | | | | | | | Yes [TC] | | | | | |
| 90 | Gandhi | 65 | F | y | | y | | | y | | | y | | | y | | | | | Y | | | Y | Y | | Y | Yes | | Yes | | | | | | | | |
| 91 | Periasamy | 56 | M | y | | | | y | | y | | | | | Y | | | | | Y | | | Y | | | | | | | | | | Yes[TC] | | | | | |
| 92 | Suresh | 41 | M | y | | | | y | | | | | | Y | | | | | Y | | | | | | Y | | | | | | | Yes | | | | | | |
| 93 | Vellaisamy | 55 | M | y | | | | | | | | | Y | | | Y | | | | | | | | | | | | | | | | | | | | | | N |
| 94 | Kamatchi | 48 | M | y | | | | y | | | | | | Y | | | | Y | | | | | Y | | | | | Yes | | | | | | | | | | |
| 95 | Poolammal | 52 | F | y | | | | | | | | | | Y | | Y | | | | | | | | | | | | | | | | | | | | | | N |
| 96 | Majith | 51 | M | y | | | | y | | | | | | | y | | | Y | | | | | Y | Y | | | | | | | | | | | | Yes | | |
| 97 | Alagar | 49 | M | y | | | | y | | | | | | Y | | | Y | | | | | | | | | | | Yes | | | | | | | | | | |
| 98 | Hariram | 72 | M | y | | y | | | y | y | | y | | Y | | | | Y | | | | | Y | | | | | | | | | Yes | | | | | | |
| 99 | Saraswathy | 67 | F | y | | | y | | y | y | | y | Y | | | Y | | | | | | | | | | | | | | | | | | | | | | N |
| ## | Ravi | 47 | M | y | | | | | | | y | | | Y | | | | Y | | | | | Y | | Y | Y | | | | | Yes[T] | | | | | | | |

**Ref. No. 23308/E4/2/2013**

**Govt. Rajaji Hospital,**
**Madurai.20. Dated: 24.12.2013**

**Institutional Review Board / Independent Ethics Committee.**
**Dr. N. Mohan, M.S., F.I.C.S., F.A.I.S.,**
Dean, Madurai Medical College &
Govt Rajaji Hospital, Madurai 625020. **Convenor**

**Sub:** Establishment-Govt. Rajaji Hospital, Madurai-20-
Ethics committee-Meeting Minutes- for December 2013
Approved list -regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held on 18.12.2013, Wednesday at 10.00 am to 12.00.noon at the Anaethesia Seminar Hall, Govt. Rajaji Hospital, Madurai. The following members of the committee have attended the meeting.

---

| | | |
|---|---|---|
| 1.Dr. V. Nagarajan, M.D., D.M (Neuro)<br>Ph: 0452-2629629<br>Cell.No 9843052029 | Professor of Neurology (Retired)<br>D.No.72, Vakkil New Street, Simmakkal, Madurai -1 | Chairman |
| 2. Dr.Mohan Prasad , M.S M.Ch<br>Cell.No.9843050822 (Oncology ) | Professor & H.O.D of Surgical Oncology(Retired)<br>D.No.72, West Avani Moola Street, Madurai -1 | Member Secretary |
| 3. Dr. I. Jeyaraj, M.S., (Anatomy)<br>Cell.No 9566211947 | Director & Professor<br>Institute of Anatomy /V.P<br>Madurai Medical College | Member |
| 4. Dr. Parameswari M.D (Pharmacology)<br>Cell.No.9994026056 | Director of Pharmacology<br>Madurai Medical College | Member |
| 5. Dr.S. Vadivel Murugan, MD.,<br>(Gen.Medicine)<br>Cell.No 9566543048 | Professor of Medicine<br>Madurai Medical College | Member |
| 6. Dr.S. Meenakshi Sundaram, MS<br>(Gen.Surgery)<br>Cell.No 9842138031 | Professor & H.O.D of Surgery i/c<br>Madurai Medical College | Member |
| 7. Mrs. Mercy Immaculate Rubalatha, M.A., Med.,<br>Cell. No. 9367792650 | 50/5, Corporation Officer's quarters, Gandhi Museum Road, Thamukam, Madurai-20 | Member |
| 8. Thiru..Pala. .Ramasamy , BA.,B.L.,<br>Cell.No 9842165127 | Advocate,<br>D.No.72.Palam Station Road, Sellur, Madurai -2 | Member |
| 9. Thiru. P.K.M. Chelliah ,B.A<br>Cell.No 9894349599 | Businessman, 21 Jawahar Street, Gandhi Nagar, Madurai-20 | Member |

The following Project was approved by the committee

..2..

| Name of P.G. | Course | Name of the Project | Remarks |
|---|---|---|---|
| Dr.P. Jeyasingh | P.G in MD., DM Cardiology, Madurai Medical College, Madurai & Government Rajaji Hospital, Madurai. | Clinical & Coronary angiographic correlation of patients with unstable angina admitted in Government Rajaji Hospital, Madurai. | Approved |

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain it Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution or to Government.
2. She/He should inform the institution Ethical Committee, in case of any change of study procedure, site and investigation or guide.
3. She/He should not deviate the area of the work for which applied for Ethical clearance. She/He should inform the IEC immediately, in case of any adverse events or Serious adverse reactions.
4. She/He should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and if any Extension of time is required He/She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the work or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

**Member Secretary** **Chairman**
**Ethical Committee**

**DEAN/Convenor**
**Govt. Rajaji Hospital,**
**Madurai- 20.**

24/12/13

**To**
**The above Applicant**
**-thro. Head of the Department concerned**

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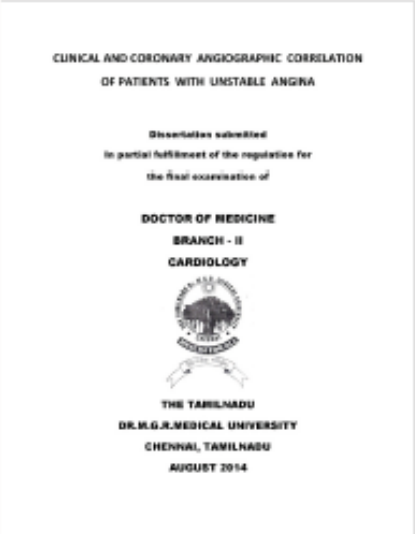

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Dissertation submitted
in partial fulfillment of the regulation for
the final examination of

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BRANCH - II
CARDIOLOGY

THE TAMILNADU
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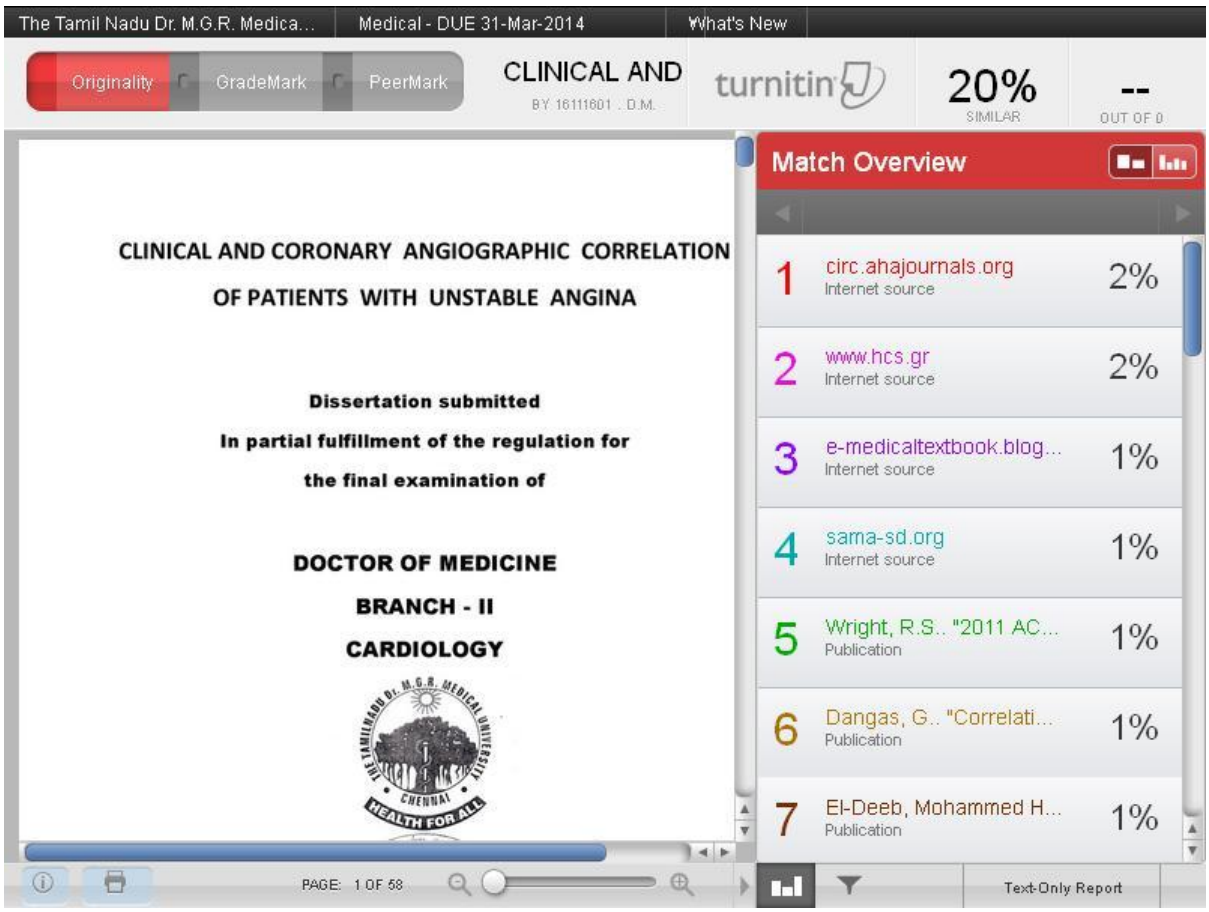
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